EXHIBIT B

Prepared for
Paul Hastings LLP

Prepared by
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EXPERT REPORT OF DR. ROSALIND SCHOOF IN THE MATTER OF

CALIFORNIA SPORTFISHING PROTECTION ALLIANCE V. PACIFIC BELL TELEPHONE COMPANY

BEFORE THE UNITED STATES DISTRICT COURT EASTERN DISTRICT OF CALIFORNIA (CASE NO. 2:21-CV-00073-MCE-JDP)

PREPARED BY

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ACRONYMS AND ABBREVIATIONS

ALM:	Adult Lead Model
ATSDR:	Agency for Toxic Substances and Disease Registry
BAF:	bioaccumulation factor
BLL:	Blood lead level
CDC:	United States Centers for Disease Control and Prevention
CSPA:	California Sportfishing Protection Alliance
DTSC:	Department of Toxic Substances
FDA:	Food and Drug Administration
IEUBK:	Integrated Exposure Uptake Biokinetic
MDL:	Method detection limit
NHANES:	United States National Health and Nutrition Examination Survey
NOAA:	National Oceanic and Atmospheric Administration
OEHHA:	Office of Environmental Health Hazard Assessment
RL:	Reporting limit
USEPA:	United States Environmental Protection Agency
WHO:	World Health Organization
µg/dL:	micrograms per deciliter
µg/L:	micrograms per liter
µg/m^2:	microgram per square meter
µg/m^3:	microgram per cubic meter
cm:	centimeter
g:	gram
kg:	kilogram
mg:	milligrams
mm:	millimeter
L:	liter
Pb:	lead

EXECUTIVE SUMMARY

I have been retained as an expert witness by Paul Hastings LLP on behalf of Pacific Bell Telephone Company (Pacific Bell) in the lawsuit captioned as *California Sportfishing Protection Alliance v. Pacific Bell Telephone Company*, in the United States District Court, Eastern District of California (Case No. 2:21-CV-00073-MCE-JDP). The complaint filed by California Sportfishing Protection Alliance (CSPA) alleges harm from two submarine lead-clad telecommunications cables formerly operated by Pacific Bell that are located in Lake Tahoe. Specifically, CSPA alleges that (i) lead dissolves from the cables and is present in Lake Tahoe water, (ii) humans ingest dissolved lead from the cables when they drink water or eat fish from the lake, and (iii) this lead exposure increases their body burden of lead and poses health risks.

My analysis documented in this report demonstrates that lead releases from the telecommunication cables are negligible and have no impact on the body burden of lead in any individuals recreating or working in or near Lake Tahoe.

Lead exposures are assessed by measuring or predicting lead levels in blood. Young children are the focus of lead health risk assessment because their potential for exposure is greater than for adults. Notably, blood lead levels in young children in the U.S. have declined 94.5 percent since the late 1970s due to regulation of historical lead sources. Blood lead levels are now so low that it is unlikely there remains any unregulated source of widespread lead exposure in the U.S.

This report sets out my detailed findings on (i) lead concentrations in water and sediment near the Lake Tahoe cables, (ii) potential exposure scenarios, and (iii) the results of applying standard methods for assessing lead exposure to quantify human health risks. In consideration of these issues, I reviewed site investigation reports, regulatory guidance, and selected scientific literature (see References for a list of information sources I considered). Based on review of these information sources, combined with my expert knowledge developed through years of education, experience, and training in toxicology and human health risk assessment, it is my opinion that the lead-clad cables in Lake Tahoe are not a lead exposure source that would trigger the preparation of a health risk assessment, because the lead concentrations involved are so low. Indeed, measured lead concentrations in water and sediment near the cables indicate that the cables are not releasing lead over their 5-6 miles of length, except potentially at locations where the cable has been cut or damaged to the point that the lead cladding is directly exposed to water, and at these places releases of lead are negligible and barely detectable.

Despite these facts showing that a health risk assessment is not necessary with regard to the cables, I applied standard methods for assessing lead exposures to quantify potential human health risks presented by the cables. When, for purposes of quantifying such health risks, I applied an assumption that people might somehow ingest water near the cut end of the submerged cable, the ingested amounts are shown to be orders of magnitude lower than typical daily lead intakes from drinking water and diet. Similarly, sediment lead concentrations near the cables are far lower than typical lead concentrations in residential soil across the U.S., and any amounts ingested will not increase body burden.

The quantitative risk analysis presented in my report confirms that, contrary to the allegations in the CSPA complaint, lead from the two submerged cables does not pose a

health risk to people, including those who use Lake Tahoe to "fish, boat, kayak, swim, bird watch, view wildlife, and engage in scientific study." Nor is there any health risk for those who "work on and in the waters of Lake Tahoe, making physical contact with those waters on a regular basis."

1. INTRODUCTION

I have been retained as an expert witness by Paul Hastings LLP on behalf of Pacific Bell Telephone Company (Pacific Bell) in the lawsuit captioned as *California Sportfishing Protection Alliance v. Pacific Bell Telephone Company*, in the United States District Court, Eastern District of California (Case No. 2:21-CV-00073-MCE-JDP). The complaint filed by California Sportfishing Protection Alliance (CSPA) alleges harm from two submarine lead-clad telecommunications cables formerly operated by Pacific Bell that are located in Lake Tahoe.

As the complaint notes, Lake Tahoe is known for its recreational activities, which include beachgoing, swimming, boating, and fishing. It is also a designated source of domestic and municipal drinking water. The complaint alleges that lead dissolves from the cables and is present in Lake Tahoe water. It then goes on to allege that *"[W]hen CSPA members contact or drink that water, their body burden of lead is increased and they, and/or their children, face a concomitant increased risk of sterility, neurodevelopmental toxicity, cancer, and other physical ailments associated with exposure to lead."* The complaint also alleges that humans ingest dissolved lead from the cables when they eat fish caught in Lake Tahoe. In contrast to these allegations, my report demonstrates that lead releases from the telecommunication cables are negligible and have no impact on the body burden of lead in any individuals recreating or working in or near Lake Tahoe.

This report provides background scientific information necessary to assess claims that releases of lead from the Lake Tahoe cables are a risk to public health, including:

- A summary of lead exposure sources and regulation,
- An explanation of how exposure pathways are identified and how exposures are measured, and
- A description of how blood lead levels are used to measure exposure.

These discussions are followed by a description of the Lake Tahoe environmental setting and my assessment of the potential for lead exposures related to the lead cladding on the cables.

Opinions discussed in this report are based on information considered at the time of report preparation. I reserve the right to modify and supplement opinions as additional information, potentially relevant to questions evaluated in this report, becomes available or is otherwise discovered.

2. QUALIFICATIONS

This section describes my qualifications to conduct this review. By education, training and experience, I am an expert in toxicology and health risk assessment. Toxicology is the scientific discipline that studies the adverse effects of chemicals on living organisms, including humans. Health risk assessment is an evaluation of the risk or hazard posed to human health by the actual or potential presence or release of hazardous substances, pollutants, or contaminants.

Since February 2010, I have been a Principal at Ramboll US Consulting, Inc. (formerly ENVIRON International Corporation). Before that, I was a Principal at several other firms. I have been board-certified by the American Board of Toxicology since 1986 (recertified in 1991, 1996, 2001, 2006, 2011, 2016, and 2021). I am a Fellow of the Academy of Toxicological Sciences and a long-time member of the Society of Toxicology (since 1998). I hold a Ph.D. in toxicology from the University of Cincinnati, as well as a B.A. in molecular biology from Wellesley College.

I have more than 30 years of experience in government and the private sector assessing human health effects and exposures from chemical substances in the environment, workplace, products, and foods. I have directed evaluations of chemical toxicity, derivation of risk-based exposure levels, health risk assessments for cancer and non-cancer end points, and multimedia assessments of exposure to environmental chemicals for diverse mining and mineral processing sites, manufacturing sites, landfills, incinerators, and community settings. I have directed numerous assessments of health risks associate with exposures to lead, including biomonitoring studies assessing potential linkages between blood lead levels and exposure sources.

I am an internationally recognized expert on the evaluation of lead and other metals in the environment and the human diet. In addition to numerous research publications on bioavailability of lead and arsenic in soil, I have developed guidance on methods for assessing the oral bioavailability of metals in soil for the U.S. Department of Defense, the Ontario (Canada) Ministry of Environment, and Health Canada.

I have served on numerous peer review panels for U.S. agencies and Canadian ministries and have been a member of three National Academy of Sciences committees. Previously, I served as a member of the Expert Advisory Panel for the Canadian Metals in the Human Environment–Research Network, the Washington Department of Ecology Model Toxics Control Act Science Panel, and the Science Advisory Panel for the Strategic Environmental Research and Development Program that funds environmental science and technology research for the U.S. Department of Defense.

I participated in several peer reviews for the U.S. Environmental Protection Agency (USEPA) related to lead, as listed below:

- 4/20—External peer review of a report summarizing an evaluation of the Integrated Exposure Uptake Biokinetic (IEUBK) Model (version 2.0), "Advancing Pb Exposure and Biokinetic Modeling for USEPA Regulatory Decisions and Site Assessments using Bunker Hill Mining and Metallurgical Complex Superfund Site Data".

- 11/14 to 2/15— External Peer Review of USEPA's Approach for Estimating Exposures and Incremental Health Effects due to Lead from Renovation, Repair, and Painting Activities in Public and Commercial Buildings.

Early in my career, I worked for the USEPA in the Office of Toxic Substances. I also worked as a toxicologist for a pharmaceutical company. A copy of my current curriculum vitae is included in Attachment 1.

3. OVERVIEW OF LEAD REGULATIONS AND REMAINING EXPOSURE SOURCES

In the U.S., lead exposures, as measured by levels of lead in the blood, peaked in the late 1970s. A series of regulations enacted since that time have resulted in markedly reduced exposures as key historical sources such as lead paint, leaded gasoline, and lead in plumbing have been reduced or controlled. Nevertheless, lower-level exposures continue due to lead naturally present in the environment and in foods, as well as remaining historical sources, and the ongoing use or presence of lead in various products or foods. Lead naturally present in the environment and foods is responsible for the low levels of lead exposure common to all people, while the remaining cases of elevated lead exposures are caused by remaining lead paint in older homes, failures of lead corrosion control in plumbing, hobbies involving lead (e.g., stained glass making or use of lead fishing tackle or bullets), and lead-contaminated products (e.g., children's toys or jewelry) and foods (e.g., spices).

In the sections that follow, I first describe the timeline of lead regulations and show how blood lead levels have declined due to those regulations. Blood lead level (BLL) measurements are most commonly used to measure human lead exposures. Lead absorbed in the human body is distributed throughout the blood and then primarily accumulated in the bone. BLLs provide an integrated measure of lead exposure from all sources.

Then I describe background exposures to lead in drinking water, soil, and food. An understanding of these background exposures is needed to assess additional potential exposures and to understand whether such exposures might result in detectable increases in lead body burden.

3.1 Timeline of Lead Regulations and Decline in Exposures

Improved understanding of lead toxicity led to various regulatory actions to reduce exposures. Increasing uses of lead caused lead exposures to reach high levels during the 1970s followed by decline as uses were reduced due to regulations. Figure 1 shows the timeline of bans on various lead uses in the United States. Despite these bans, the presence of lead in the environment and in older homes remains pervasive throughout the U.S. due to these historical uses. For example, the addition of lead as an antiknock compound in gasoline for 50 years has resulted in widespread elevation of lead concentrations in soils near roadways (ATSDR 2020). Indeed, this source is proposed as a primary source of the lead reported in deep sediments of Lake Tahoe (Hayvaert 2000).

Figure 1 also provides an overview of the decline in BLLs in the U.S. as various regulations were implemented. Lead exposure assessment is generally based either on estimated daily intakes in micrograms (µg) or on measurement or prediction of blood lead levels.

In developed countries, BLLs have declined markedly over the past 50 years. Much of the decrease in BLLs can be attributed to the ban of leaded gasoline, ban of lead-based paint use in residential buildings, and improvements in corrosion control for drinking water systems. Young children are generally the focus of assessments of lead exposures because they have historically exhibited the highest blood lead levels. As of 2016, average blood lead levels in young children had declined 94.5% from those in 1976–1980. Average (geometric mean) blood lead declined from 15.2 micrograms per deciliter in the period from 1976–1980 to 0.83

micrograms per deciliter in the period from 2011–2016 (Figure 2; Egan et al. 2021 Env Health Perspectives). This remarkable and pervasive decline in blood lead levels would not have been possible if there was an unknown significant nationwide source of lead contamination to which children were being exposed.

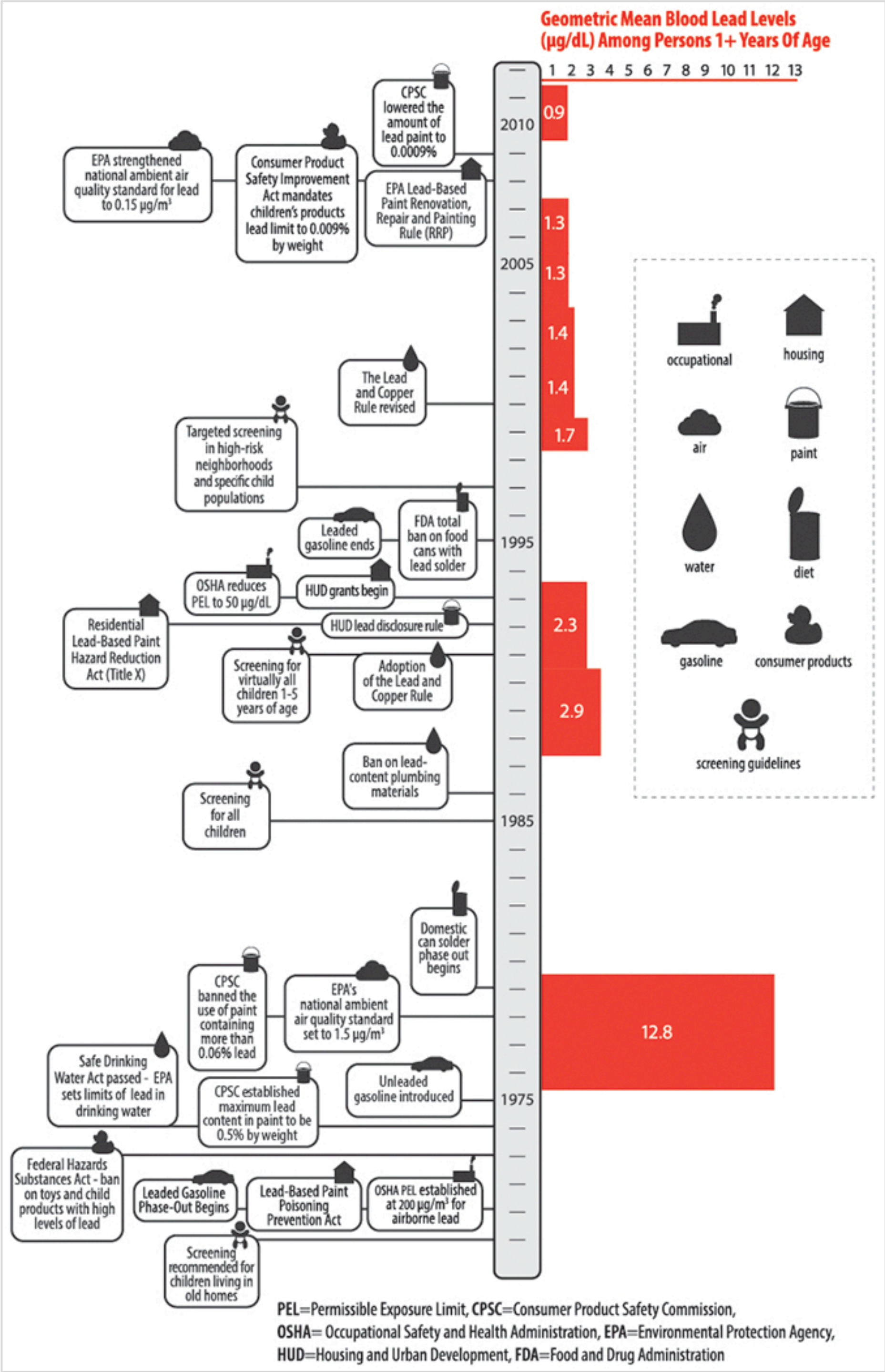

Figure 1. Timeline of lead regulatory events in the U.S. (From: Dignam et al. 2019)

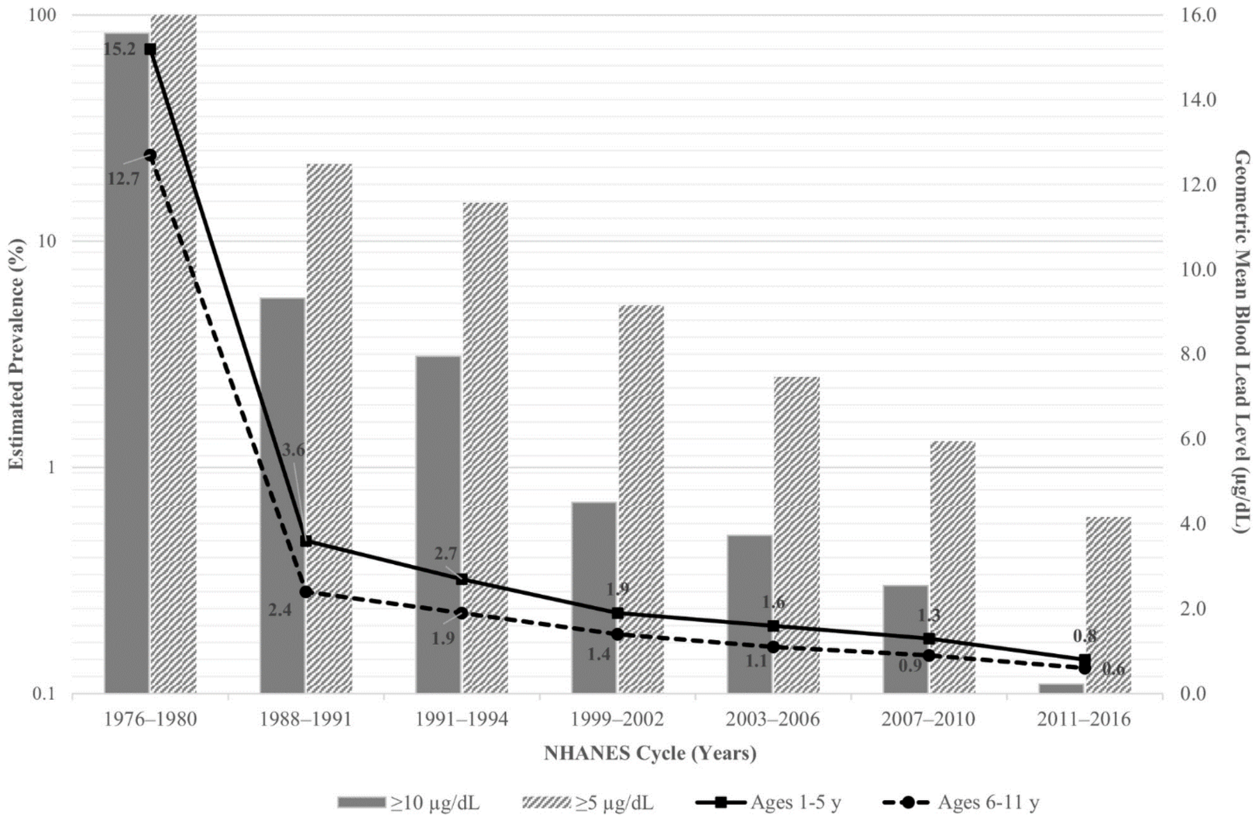

Figure 2. Decline in Geometric Mean Blood Lead Level 1976–2016 (From: Egan et al. 2021)

Decline in geometric mean blood lead levels and prevalence of blood lead levels ≥10μg/dL (gray bars) and ≥5μg/dL (hatched bars) 1976–2016, U.S. children 1–11 years

3.2 Lead Exposure Sources

Lead can be found in all parts of our environment, including water, soil, and air, and in foods we eat, meaning that everyone has some lead in their blood. Low levels of lead are present naturally, but anthropogenic activities have frequently resulted in higher concentrations in drinking water and in soil. Intake from the diet can be the greatest source of background exposure when intakes from drinking water and soil are low.

3.2.1 Drinking Water Systems

Lead in drinking water can derive from source water contamination, but more commonly originates from internal corrosion of water distribution system piping and plumbing (ATSDR 2020). Background lead concentrations in U.S. drinking water has been reported in a number of studies, as well as published by federal organizations. Bradham et. al (2022) reviewed 678 drinking water samples from the American Healthy Homes Survey II and found that the mean concentration was 1 μg Pb/L. USEPA currently uses a background water lead concentration of 0.9 μg/L for lead risk assessment for children. This value was derived from

the 1998-2005 Six-Year Review-Information Collection Request (ICR) Dataset[1], a comprehensive assessment of drinking water quality and water treatment capabilities (USEPA 2010).

Intakes of lead from drinking water will vary based on how much tap water each individual drinks. A child who ingests one liter of drinking water per day containing lead at the average concentration (1 µg/L) will ingest about 1 µg of lead per day.

3.2.2 Soil

Geologic background for lead in soil is typically in the range of 10 to 30 mg/kg (Shacklette and Boerngen 1984), but soil lead levels in many urban areas exceed 200 mg/kg (AAP 1993). The most recent American Health Homes Survey (AHHS II; HUD 2021) reported a mean soil lead concentration of 106 mg/kg for residential areas in the United States. Average estimates varied by region, as high as 222 mg/kg in the Northeast to 53 mg/kg in the South. A meta-analysis of soil concentrations by USEPA researchers (Frank et al. 2019) estimated a mean soil lead concentration of 629 mg/kg in urbanized areas and 284 mg/kg in non-urbanized residential areas. The units of mg lead per kg soil are the same as µg lead per gram (g) of soil.

People are thought to ingest very small amounts of soil (referred to as incidental soil ingestion) due to hand-to-mouth activities when hands are not clean. Children have much greater hand-to-mouth activity than adults and are expected to have greater rates of incidental soil ingestion. For example, a 2-year-old child might ingest 67 milligrams (mg)[2] of soil per day; 67 mg of soil is the same as 0.067 g of soil. If a child ingests 0.067 g of soil that has 100 µg/g lead, that child would ingest 6.7 µg of lead.

3.2.3 Air

Lead concentrations in ambient air are collected from four national monitoring networks that are reported to the USEPA's Air Quality System. Between 2008 and 2010, the measured source-oriented and nonsource-oriented monthly median lead concentrations were 0.063 µg/m^3 and 0.010 µg/m^3, respectively (ATSDR 2020, USEPA 2014).

If a child breathes 10 m^3 of air per day of air with 0.010 µg/m^3, they will inhale 0.1 µg of lead.

3.2.4 Food

Lead may be present in food because it is in the environment where foods are grown, raised, or processed. The Food and Drug Administration (FDA) detected lead in 15 percent of foods sampled during the 2018-2020 FDA total diet study, a nationally representative sample of commercially available foods commonly consumed in the United States (FDA 2022a). Foods with the highest mean lead concentrations were baby food sweet potatoes (21 µg/kg), baby food teething biscuits (18 µg/kg), sandwich cookies (13 µg/kg), white wine (13 µg/kg), and low-calorie ranch salad dressing (13 µg/kg) (FDA 2022b).

Average dietary lead intake estimates for children (1–6 years) in the United States vary, ranging from roughly 1 to 6 µg/day, depending on the age of child, data source, and analysis method used. The USEPA has estimated daily dietary lead intakes of roughly 5-6 µg/day for

[1] The default value was derived as a population-weighted, average estimate of high-end exposure data (USEPA OLEM n.d.(a)).

[2] Default assumption from EPA's IEUBK model.

use in the Integrated Exposure Uptake Biokinetic Model (USEPA OLEM n.d.(b)). A separate analysis from the USEPA Office of Research and Development (Zartarian et al. 2017), which uses more recent dietary consumption and dietary lead concentration data, estimates daily lead intakes of roughly 2-3.3 µg/day. An analysis from the FDA (Spungen 2019) estimated mean dietary lead exposures ranging from 1.2 to 3.2 µg/day.

3.2.5 Impact of Lead in Drinking Water, Diet, Soil and Air on Children

Table 1 summarizes estimated background lead intakes in µg/day for a young child, the age group most vulnerable to lead exposures. For this age group, the majority of background lead exposure comes from diet and soil, followed by drinking water, with very little from air. Intake from soil is uncertain and highly variable depending on soil lead concentrations present and frequency and intensity of contact. The values in Table 1 reflect potential intakes, not absorbed doses. Absorbed dose would be lower for all media and is influenced by media-specific characteristics as well as individual-specific characteristics such as nutritional status (ATSDR 2020). Much less lead is absorbed from ingested soil than from drinking water and food. The absorbed dose associated with lead intake from soil would be proportionally lower than the intakes shown in Table 1.

Table 1. Background Lead Intakes (µg/day)

Media	Two-year-old child
Drinking water (1 µg/L)	0.6–1.0
Diet	1–6
Air (0.01 µg/m^3)	0.1
Soil (including house dust) (50–200 mg/kg)	3–14

3.2.6 Other Sources of Lead

In addition to natural sources of lead in water, soil, air and foods, multiple historical uses of lead in paint, gasoline, plumbing and various consumer products have contributed to historical and ongoing lead exposures. Household products to which children could be exposed include imported cosmetics and traditional medicines, imported toys or jewelry containing lead or coated with lead-based paints, and imported foods such as candies, spices, and canned food from countries where lead solder is still used (FDA 2024). Lead can be leached from tea kettles, lead-glazed dishes and lead crystal decanters and glassware. Vinyl miniblinds containing lead salts to reduce degradation of the plastic have been found to contaminate house dust. Children and adults have also been exposed to lead when they assist in handling lead-containing ammunition or fishing tackle, recycling lead batteries, or working with lead solder when working with stained glass (Demmeler et al. 2008; Kaul et al. 1999).

4. EXPOSURE ASSESSMENT METHODOLOGY

Exposure assessment is one step of the process used to assess human health risks. For most chemicals, an exposure assessment yields an exposure measure usually stated in terms of a daily dose (for ingestion or dermal exposures) or an air concentration (for inhalation exposures). These exposure doses or concentrations are then compared with toxicity values that represent doses below which there is no risk of adverse effects. If the exposure doses or concentrations are higher than the toxicity values, further analysis will determine if any risk exists.

Regulatory agencies, including the USEPA, use exposure assessment and exposure pathway models to interpret chemical sampling results from environmental media and calculate exposure doses. Exposure pathway models (often referred to as conceptual site models) include analyses of the sources of chemicals and how chemicals move in the environment. An exposure pathway model will tell you if there is a way for released chemicals to reach people, flora and fauna, and if so, what that pathway is. If the released chemicals cannot contact people, flora or fauna, there is no potential for adverse effects. Such incomplete exposure pathways can occur due to breaks at any point in this “chain” connecting a source to the receptor. For example, if lead is released from a buried cable, but is not transported to surface soil, the exposure pathway for people is likely incomplete.

An exposure pathway model should be used to guide data collection, interpretation of results, and development of plans for additional studies to determine if complete exposure pathways exist. Assertions that lead measured in the environment is causing exposure or adverse health effects must be linked to a full exposure pathway analysis. For example, water concentrations in areas where children could readily swim must be measured or modeled before such assertions are made. Mismatches between environmental concentrations and areas where people may be exposed may also occur. For example, it would not be reasonable to assume that a child plays all day, every day in soil in a planting strip next to a busy street. Lead concentrations in soil in the planting strip should not be taken as an indication of risk to children using exposure models based on residential yards.

Even if a complete exposure pathway is thought to exist, an unacceptable risk is not necessarily present. A risk assessment must be conducted to calculate potential exposures and determine if risk thresholds may be exceeded. Risk assessments usually include conservative assumptions that ensure risk estimates will protect the most susceptible people and may overestimate risks for most people.

Exposures for complete exposure pathways are estimated by combining information about chemical concentrations in an environmental medium (e.g., water, soil, air), how often someone contacts the medium, and other factors related to duration of contact, body weight, and life-stage of the population. Exposures can be estimated as the amount of a chemical ingested or inhaled by a person per day or the amount ingested or inhaled can be estimated per unit body weight. For example, water concentration in milligrams (mg) of chemical per liter (L) of water (mg/L) multiplied by the liters drunk per day, multiplied by the number of days or fraction of days drinking the water, divided by total days per year and body weight in kilograms (kg), yields a dose in terms of mg/kg-body weight. Assumptions for exposure parameters, such as the amount of water ingested each day, are based on literature sources.

The USEPA Exposure Factors Handbook provides a compilation and recommendations for many exposure parameters.

In the U.S., a modified approach is used to assess exposures. Lead exposure in terms of daily intake (in units of µg/day) from various sources is calculated based on average values for various exposure parameters in a similar manner as for other chemicals. These intakes are then input into a toxicokinetic model to predict a range of blood lead levels associated with the exposure. As noted above, integrated lead exposure from multiple sources is assessed using BLLs, in units of micrograms of lead per deciliter of blood (µg/dL). Most public health benchmarks for lead are expressed as BLLs. Common models used by USEPA and the California Office of Environmental Health Hazard Assessment (OEHHA) to predict blood lead concentrations from exposure to lead in environmental media include USEPA's Integrated Exposure Uptake Biokinetic Model (IEUBK), USEPA's Adult Lead Model, and California Department of Toxic Substances' (DTSC) LeadSpread model.

5. BLOOD LEAD AS AN INDICATOR OF LEAD EXPOSURE

As discussed above, BLLs provide an indication of lead exposures. The following section describes how lead behaves in the body once absorbed, how blood lead is measured and sources and factors affecting BLLs. The last section describes blood lead reference and guideline values.

5.1 Blood Lead Reflects All Recent Exposures

Concentrations of lead in the blood are used to assess recent lead exposure from all sources. BLLs are stated in units of micrograms per deciliter (µg/dL); a deciliter refers to 1/10th of a liter or 0.1 liters. Blood lead measurements are dependent on the lapse of time between the measurement and the lead exposure, and generally reflect a combination of exposure to lead over the past 1–3 months plus the influence of any lead released from bone during that time. Following ingestion, absorbed lead is widely distributed to blood plasma and soft tissues, and is accumulated in bone (ATSDR 2020). Blood typically only contains a small fraction of the total body burden relative to bone.

The half-life of lead in blood is estimated to be about 30 days for adults (ATSDR 2020), meaning that if there is no continuing exposure to lead, the concentration in the blood would decline to half the starting value in about 30 days. During that time some of the lead would be excreted in the feces and urine and some would be stored in bone. The half-life of lead in bone ranges from 10–30 years in adults (NTP 2012), reflecting the long-term storage in bone. Lead may be mobilized from bone by any physiological state that causes bone to remodel. This includes growth of children and adolescents, pregnancy, and osteoporosis. Thus, while blood lead generally reflects recent lead exposures, it may also include a contribution from historical exposures if stored lead is released from bone. In children, a smaller proportion of the body burden of lead is stored in bone (ATSDR 2020). Continuous growth in children results in constant bone remodeling, and bone lead is exchanged with blood lead much more frequently than in adults (NTP 2012).

Blood samples may be collected by several methods, including "finger stick," with blood collection into a capillary tube, or venous samples with blood collected using a needle and syringe system. Venous samples are considered to provide more reliable data than finger stick samples (NHMRC 2016), which may have elevated lead levels due to external contamination from lead on the skin. Analytical methods vary with regard to analytical detection limits. A common lead surveillance method (LeadCare II) has a detection limit of 3.3 µg/L, much higher than laboratory analytical methods (with detection limits of 1 µg/L or lower). The reliability of reported blood lead data depends on the collection and analytical methods.

BLLs are generally higher in young children than in adults because young children may absorb lead more efficiently and because they have more hand-to-mouth activity, causing them to ingest more lead from soil, dust, and household objects.

Many factors are known to be correlated with higher BLLs. Multiple studies have shown that, on average, children residing in older homes have higher BLLs, especially for homes built prior to the ban of lead-based paint use in residential buildings (HUD 2021, Kim et al. 2002). Houses built before 1978 are likely to contain lead-based paint, and houses built prior to 1940 may have used paint that contained 50 percent lead, the average for interior paints at the time (Dignam et al. 2019). The presence of peeling paint in the home is also associated

with higher BLLs. Demographic factors that have an influence on BLLs of children include level of education and socioeconomic status of parents (Nriagu et al. 2006). These factors may be interlinked and more likely to be found in households below the poverty line.

BLLs are known to exhibit a seasonal effect, with higher numbers of elevated BLLs measured in the summer, and lower numbers of elevated BLLs measured in the winter months. This is often attributed to higher exposure to lead in soil and dust due to increased outdoor activity, among other factors (Laidlaw et al. 2016, Haley and Talbot 2004). Water lead may also contribute to seasonality because warmer temperatures decrease pH and increase lead solubility in water. Ngueta (2014) found the change from winter to summer climate can cause average BLLs to rise by 1 µg/dL.

5.2 Blood Lead Reference and Guideline Values

Available studies have not yielded a clear threshold dose or BLL below which we can confidently state lead has no adverse effects. Historically BLLs were too high to provide enough data to assess lower-level exposures. More recent studies suffer from methodological limits that prevent clear definition of a threshold level. Given this uncertainty, in 2012 the Centers for Disease Control and Prevention (CDC) began using a population-based reference concentration as a marker of elevated BLLs in children. The derivation of this value is described below, followed by a description of an alternate approach used by the State of California, and a brief discussion of the typical causes of elevated BLLs in U.S. children.

5.2.1 CDC Blood Lead Reference Values

As BLLs have declined over time, so has the concept of an elevated blood lead level. In 1970, BLLs >40 µg/dL were considered elevated. From the mid-70s to the mid-80s, BLLs >30 µg/dL were considered elevated. This number dropped to 25 µg/dL from the mid-80s until 1991, when the CDC defined BLLs >10 µg/dL as elevated (CDC 1991). In 2012, CDC recommended that the elevated BLL definition for children be revised to BLLs >5 µg/dL. BLL surveillance conducted as a part of the National Health and Nutrition Examination Survey (NHANES) has played a large role in defining elevated BLLs. The 5 µg/dL reference level was based on the 97.5th percentile BLL measured in children aged 1–5 years in the 2007–2010 NHANES data sets, i.e., this is a reference value based on population statistics and is not an indication of a threshold for adverse health effects. As BLLs have continued to decline, CDC re-evaluated the NHANES data and in 2021 identified a new reference BLL of 3.5 µg/dL based on the 97.5th percentile BLL measured in children aged 1–5 years in the NHANES data from the 2015–2016 and 2017–2018 cycles.

The CDC reference values are used by state health departments to identify children who have elevated BLLs so that their families can be provided guidance on reducing exposures.

5.2.2 California Blood Lead Health Guidance Value

In 2007 OEHHA developed a 1 µg/dL health guidance value (HGV) for source-specific incremental change in blood lead levels for protection of school children and fetuses. According to OEHHA, 1 µg/dL is the estimated incremental increase in children’s blood lead that would reduce IQ by up to 1 point (OEHHA 2007, 2022). The OEHHA HGV approach differs from the reference value approach currently used by CDC because the 1 µg/dL benchmark corresponds to a specific health effect (reduced IQ by up to 1 point) instead of a reference level BLL (e.g., 97.5th percentile) in the population. The analysis conducted by OEHHA relies on a study (Lanphear et al. 2005) that has been shown to have uncontrolled

confounding, rendering their conclusions about potential IQ effects highly uncertain (Van Landingham et al. 2020).

5.2.3 Typical Causes of Elevated Blood Lead Levels

Elevated BLLs can result from exposure to a wide variety of lead sources that may be present in the home, such as those described in Section 3.2. When a child with a markedly elevated BLL is discovered, representatives of health authorities typically conduct a home assessment to identify the specific causes responsible for that child (CDC 2002, ATSDR 2017). As shown in a U.S. study, the most common source of significantly elevated BLLs is deteriorated lead paint (Levin et al. 2008), but elevated BLLs in children are also frequently caused by lead painted toys, traditional remedies, pottery, and numerous other sources.

The most effective way to reduce lead exposure in all children is to focus on reducing exposure to deteriorated lead paint and other known sources in the home. Numerous studies have shown that blood lead levels are highest in low-income children living in older homes and rental homes. Dignam et al. (2019 J Public Health Manag Pract) discuss control of lead sources and conclude "*Approximately 23 million housing units contain significant lead-based paint hazards, and 6.1 million have lead water service lines. Low-income, poorly maintained rental properties may be the first priority to eliminate or control these hazards, but resources are also needed for single-family-owned properties*." Levin et al. (2008) estimate that 70% of elevated BLLs in children are due to lead from paint.

6. ASSESSMENT OF POTENTIAL LEAD RELEASE FROM SUBMARINE CABLES

As discussed above, an exposure pathway model begins with characterizing the source of chemicals and how chemicals move in the environment. In this section, I describe the Lake Tahoe lead-clad cables and provide an analysis of their potential to release lead to the environment based on data collected in water and sediment near the cables. Finally, I show that the measured lead concentrations are not elevated above background levels of lead in Lake Tahoe's surface water and sediment.

6.1 The Design of the Submarine Cables Limits Lead Releases

The submarine cables at issue in Lake Tahoe contain an internal layer of lead to protect the interior copper telecommunications wiring. The lead cladding is then covered by a steel jacket and jute cover that limit release of lead from the lead sheath to surface water. Any leached lead is expected to form relatively insoluble precipitates in water and these precipitates will sorb to suspended particles that will be deposited in sediment near the cable. Most water bodies will have massive dilution potential relative to the rate of lead release from any portion of a cable where the lead sheath is exposed. Consequently, the potential for surface water contamination at any distance from a submarine cable is expected to be negligible. Similarly, any measurable impacts to sediment are expected to be limited to sediment below portions of cable where the lead sheath is exposed, or immediately surrounding the cable if buried in sediment.

6.2 Lake Tahoe Data Show Negligible Lead Concentrations Near the Cables

Water and sediment samples have been collected near two lead-clad cables in Lake Tahoe. Cable locations are shown in Figure 3. One cable (Cable A) stretches along the lake bed across the mouth of Emerald Bay. The second cable (Cable B) runs generally from north to south along the lake bed in shallow water near the western shore of the lake. Cable B extends more than four miles north of the mouth of Emerald Bay and several miles south to the northwest end of Baldwin Beach. The lead sheathing is intact for all of Cable B, and the majority of Cable A (Ramboll 2023b). Cable A is cut in one location at the north end of Emerald Bay, and the lead sheath and copper wire core are exposed. Additionally, the terminus end of Cable A is cut off and sealed with a plastic container. The depths of the cables vary, from greater than 10 feet to less than 4 feet underwater. Cable B is located in sediment on shore in one location (Baldwin Beach). In 2021, Hayley & Aldrich collected water samples. During 2023, Ramboll collected samples of water and sediment.

The results of the Ramboll and Haley & Aldrich studies are described below. Both studies demonstrate little to no release of lead from either cable. In fact, the results are among the lowest water and sediment lead concentrations of any study I have reviewed.

PROJECT: 1940106016 | DATED: 4/1/2024 | DESIGNER: CWILLIAMSFREIER C:\Users\CWILLIAMSFREIER\Ramboll\Melody Kneale - Projects\AT&T\03_GIS\20240118_SedimentSampling\20240118_SedimentSampling.aprx\Figure 6-1 - Locations of Cable A and Cable B in Lake Tahoe

LEGEND

Cable Lines

0 1 2 Miles

LOCATIONS OF CABLE A AND CABLE B IN LAKE TAHOE

Lake Tahoe
California

FIGURE 3

RAMBOLL AMERICAS
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6.2.1 Haley & Aldrich 2021 Surface Water Study

The study conducted by Haley & Aldrich in 2021 found that the cables are not adversely impacting the lake's water quality and that any detected lead was consistent with background levels of lead in the lake.

Haley & Aldrich collected water samples from five stations, including one near Cable A (Station 1) and one near Cable B (Station 2), and three control stations (Stations 3 through 5) (see Figure 4). Station 1 is located at the eastern cut end of Cable A where the underlying lead sheathing directly contacts lake water. Per Haley & Aldrich (2021), the location of Station 2 was determined following video survey conducted on Cable B spanning the entrance to Emerald Bay. The three control stations are located at varied distances from the cables (Station 3 is 100 feet (ft) northeast of Station 1; Station 4 is 600 ft northeast of Station 1; and Station 5 is near the Tahoe Keys area and 3.5 miles southeast of Stations 1 and 2.)

At Stations 1 and 2, samples were collected at varying distances from the cable, including 4 inches from each cable at cable depth; 6 inches above each cable (duplicates were collected); midway between lake surface and lake bottom; and approximately 6 inches below the lake surface. At Stations 3 through 5, the lake samples were collected 1 foot above the lake bottom. Two additional samples were also collected at Station 5; one midway between the lake surface and lake bottom; and one 6 inches below the lake surface.

The water samples were analyzed for dissolved and total lead. As shown in Figure 4, lead was not detected in the majority of the Haley & Aldrich samples. Of the few samples where lead was detected (located at Stations 1, 2, and 5), the concentrations were very low and just slightly above the method detection limit of 0.043 µg/L but well below the method reporting limit of 0.3 µg/L[3].

As a result of these findings, Haley & Aldrich concluded that the lake's water quality is not adversely impacted by the two cables (Cable A and Cable B). Lead was not detected in the majority of the water samples. The few samples where lead was detected reported very low concentrations that were just above the method detection limit, "and within the same range regardless of proximity to the subject cables" (Haley & Aldrich 2021). Haley & Aldrich noted that the low lead concentrations more closely resemble those of background levels (i.e., lead levels investigated in Chien et al. [2019]) than concentrations associated with releases from the cables.

[3] The method detection limit (MDL) is the minimum measured concentration of lead in water that can be detected and measured with a 99% confidence that the concentration can be distinguished from method blank results. A reporting limit (RL) is the lowest measurable concentration within limits of precision during standard laboratory operations.

Notes:
1. Source – Haley & Aldrich, 2024

Haley & Aldrich 2021 Surface Water Sampling Results

FIGURE 4

Lake Tahoe
California

RAMBOLL AMERICAS
ENGINEERING SOLUTIONS, INC.
A RAMBOLL COMPANY

6.2.2 Ramboll 2023 Surface Water Study

In June 2023, Ramboll sampled surface water in the vicinity of the two lead-clad telecommunication cables in the lake. Ramboll collected water samples at twelve (12) locations. This included six stations near Cables A and B (Stations 1 through 6), six reference locations, and a single offshore location (Figure 5). Of the six reference locations, three were based on sample location coordinates from the Haley & Aldrich study (2021).[4] Sampling depths at each station are noted in Table 2.

The water samples were analyzed for dissolved and total lead. The method detection limit (MDL) for lead in the Ramboll study was 0.006 µg/L, meaning lead could be detected at lower concentrations than in the Hayley & Aldrich study. The majority of samples collected at Stations 1 through 6 (i.e., near the cables) had total and dissolved lead concentrations reported either below the method reporting limit (MRL) of 0.02 µg/L or below the MDL of 0.006 µg/L. The majority of reported results were "J" flagged, indicating an estimated value where lead was present, but the actual concentration was estimated because it is below the laboratory reporting limit. Only Station 4, which is located near a cut portion of Cable A, had lead concentrations above the reporting limit (0.027–0.049 µg/L dissolved lead; 0.044–0.067 µg/L total lead).

Similarly, lead was either below the reporting limit or not detected in the reference samples. Total lead at the Reference 2 location was between the MRL and the MDL (i.e., "J" flagged). Two of the 3 reference samples sharing the same coordinates as those in the Haley & Aldrich study (HA Reference 1 and 2) were also J-flag estimated concentrations. Lead was not detected in the single offshore sample.

The 2023 Ramboll surface water results are consistent with the findings of Haley & Aldrich in 2021: both studies found lead only at very low levels (i.e., less than 0.1 µg/L) in Lake Tahoe, even when sampled directly next to the cables.

[4] The study performed by Haley & Aldrich (2021) used similar sample collection methods to those used in the Ramboll (2023) study. Both collected water samples less than twelve inches above the cable and utilized similar equipment (Van Dorn vs Kemmerer). Key differences included:

- The coordinates, especially for Station 5, varied between studies but both located the cable in the same area and similarly collected the sample; and
- The laboratory reporting limits differed: Haley & Aldrich MDL = 0.043 µg/L; Ramboll MDL = 0.006 µg/L. As a result, the Ramboll study was able to detect minute increases in lead concentrations near the cut end of the cable that could not be detected with the higher MDL from the Haley & Aldrich study.

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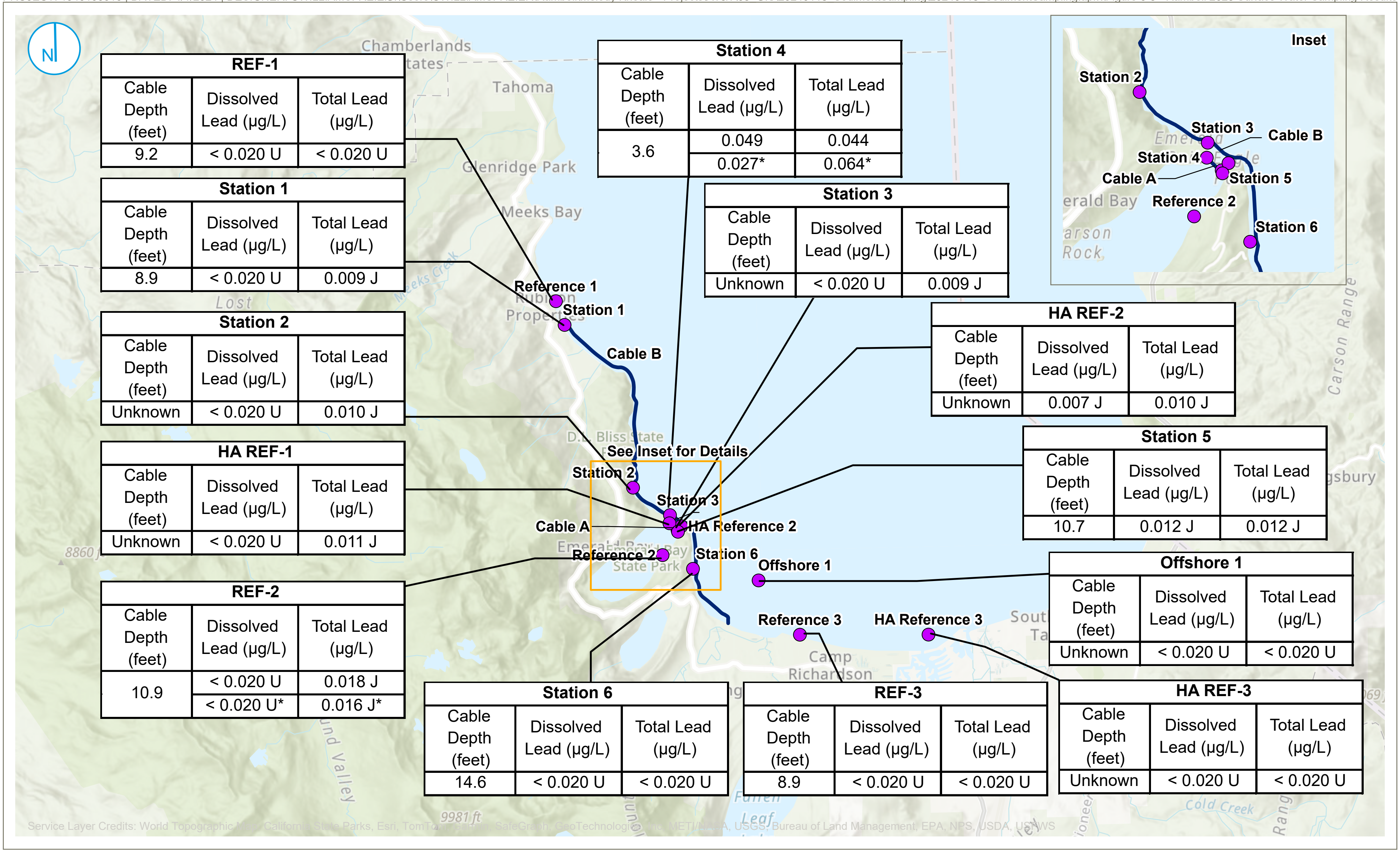

REF-1

Cable Depth (feet)	Dissolved Lead (µg/L)	Total Lead (µg/L)
9.2	< 0.020 U	< 0.020 U

Station 1

Cable Depth (feet)	Dissolved Lead (µg/L)	Total Lead (µg/L)
8.9	< 0.020 U	0.009 J

Station 2

Cable Depth (feet)	Dissolved Lead (µg/L)	Total Lead (µg/L)
Unknown	< 0.020 U	0.010 J

HA REF-1

Cable Depth (feet)	Dissolved Lead (µg/L)	Total Lead (µg/L)
Unknown	< 0.020 U	0.011 J

REF-2

Cable Depth (feet)	Dissolved Lead (µg/L)	Total Lead (µg/L)
10.9	< 0.020 U	0.018 J
	< 0.020 U*	0.016 J*

Station 4

Cable Depth (feet)	Dissolved Lead (µg/L)	Total Lead (µg/L)
3.6	0.049	0.044
	0.027*	0.064*

Station 3

Cable Depth (feet)	Dissolved Lead (µg/L)	Total Lead (µg/L)
Unknown	< 0.020 U	0.009 J

HA REF-2

Cable Depth (feet)	Dissolved Lead (µg/L)	Total Lead (µg/L)
Unknown	0.007 J	0.010 J

Station 5

Cable Depth (feet)	Dissolved Lead (µg/L)	Total Lead (µg/L)
10.7	0.012 J	0.012 J

Offshore 1

Cable Depth (feet)	Dissolved Lead (µg/L)	Total Lead (µg/L)
Unknown	< 0.020 U	< 0.020 U

Station 6

Cable Depth (feet)	Dissolved Lead (µg/L)	Total Lead (µg/L)
14.6	< 0.020 U	< 0.020 U

REF-3

Cable Depth (feet)	Dissolved Lead (µg/L)	Total Lead (µg/L)
8.9	< 0.020 U	< 0.020 U

HA REF-3

Cable Depth (feet)	Dissolved Lead (µg/L)	Total Lead (µg/L)
Unknown	< 0.020 U	< 0.020 U

LEGEND

- Water Sampling Location
- Cable Lines

Notes

1. All samples were collected in July, 2023
2. * = field duplicate sample
3. µg/L = micrograms per liter
4. U = not detected above laboratory reporting limits
5. J = estimated concentrations below the laboratory reporting limit and the laboratory detection limit

0 1 2 Miles

RAMBOLL 2023 SURFACE WATER SAMPLING RESULTS

Lake Tahoe
California

FIGURE 5

RAMBOLL AMERICAS ENGINEERING SOLUTIONS, INC.
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Table 2. Ramboll Study Results for Water Sampling (2023)

Sample ID	Dissolved Lead (µg/L)	Total Lead (µg/L)	Sampling Depth [a]
Station 1-01-062023-C	Not detected	0.009 J	8’9”
Station 2-01-062023-C	Not detected	0.010 J	9’8”
Station 3-01-062023-C	Not detected	0.009 J	11’10”
Station 4-01-062123-C	**0.049**	**0.044**	2’9”
Station 4-DUP-062123-C*	**0.027**	**0.064**	2’9”
Station 5-01-062123-C	0.012 J	0.012 J	10’
Station 6-01-062123-C	Not detected	Not detected	4’11”
Offshore 1-01-062123-NC	Not detected	Not detected	20’
Reference 1-01-062023-NC	Not detected	Not detected	9'4"
Reference 2-01-062023-NC	Not detected	0.018 J	15'9"
Reference 2-DUP-062023-NC*	Not detected	0.016 J	15'9"
Reference 3-01-062023-NC	Not detected	Not detected	7'11"
HA Reference 1-01-062123-NC	Not detected	0.011 J	11'5"
HA Reference 2-01-062123-NC	0.007 J	0.010 J	13'9"
HA Reference 3-01-062123-NC	Not detected	Not detected	11'

Notes:

µg/L – micrograms per liter

J – reported as an estimated value

* – indicates field duplicate.
’ = feet
” = inches

Bold – indicates measured value

[a] = approximate depth
“Not detected” means the analyte was not detected at or above the MDL of 0.006 µg/L

6.2.3 Ramboll 2023 Sediment Study

In July 2023, Ramboll undertook a sediment investigation to determine the potential for Cable A and Cable B to contribute to lead concentrations in sediments (Ramboll 2023b). Sediment samples were collected near both cables, at reference locations, and at publicly accessible beach areas. Results of this study determined that lead concentrations in nearshore Lake Tahoe sediments were very low at all locations sampled.

Samples were collected from twelve locations, including five cable stations (three at Cable A and two at Cable B); three reference stations; and four beach stations. Locations of these samples are shown in Figure 6.

Of the five cable station sampling locations, three were located adjacent to Cable A (SED4, SED5, and SED5-END) and two adjacent to Cable B (SED1, SED6). Sediment was sampled at three distances from the cable: within 15 centimeters (cm), 1 meter (m), and 2 m. The cores closest to the cable were designated “A” samples, the cores at 1 m were designated “B” samples, and the cores at 2 m were designated “C” samples.

All three reference stations (REF1, REF2, and REF3) were positioned at the same locations as were used in the water study (see Section 6.2.2). REF1 was more than one-half mile north of the end of Cable B and located at water depth of approximately 9 feet 2 inches. Samples at REF2 were characterized as hard sand over rocky substrate. REF3 was located along the southern shoreline of the lake.

In addition to the cable and reference stations, samples were also collected from four beach stations (Meeks Bay, Emerald Bay, Baldwin Beach, and Pope Beach), which are considered reference locations as well. The sediment samples were collected from the bathing/wading area of public beach access areas in Lake Tahoe. Only one end of Baldwin Beach has a cable present, and the sample was not collected near the cable.

As shown in Table 3, lead concentrations collected closest to all cables (e.g., within 15 cm of the cable) ranged from 0.659 to 7.57 mg/kg[5]. Lead concentrations at reference locations ranged from 0.548 to 2.45 mg/kg, while concentrations at beach sites ranged from 0.920 to 1.4 mg/kg. The highest lead concentrations were measured in the samples closest to where Cable A had been cut (SED4 and SED5-END). Lead concentrations directly below the cut/exposed cable at station SED4 averaged 5.4 mg/kg while lead one meter away was 2.0 mg/kg. At SED5-END, where the cable had been cut and capped with a sealed plastic container, the lead concentration in sediment was 5.57 mg/kg directly beneath the cable and 1.77 mg/kg one meter away.

The average lead concentration across all samples located closest to the cables (“A” samples) was 2.72 mg/kg. Average lead concentrations for the “B” (1 m) and “C” (2 m) samples were 1.39 mg/kg and 1.11, respectively. The reference stations had average lead concentrations of 1.25 mg/kg, while beach stations within the wading/bathing zone had average lead concentrations of 1.22 mg/kg.

[5] The sample with a concentration of 7.57 was a duplicate. The concentration reported in the primary sample was 3.33.

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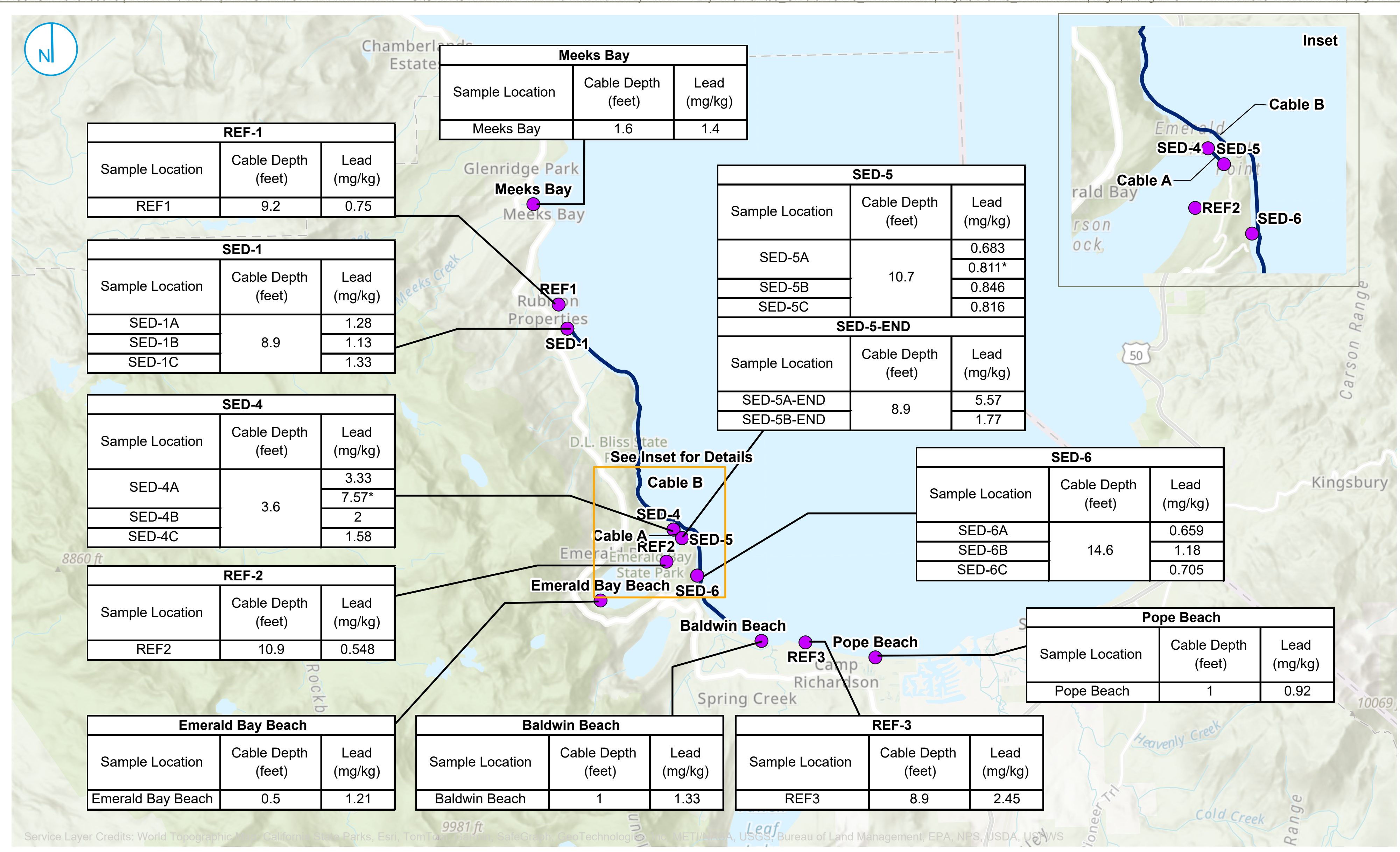

Meeks Bay

Sample Location	Cable Depth (feet)	Lead (mg/kg)
Meeks Bay	1.6	1.4

REF-1

Sample Location	Cable Depth (feet)	Lead (mg/kg)
REF1	9.2	0.75

SED-1

Sample Location	Cable Depth (feet)	Lead (mg/kg)
SED-1A	8.9	1.28
SED-1B		1.13
SED-1C		1.33

SED-4

Sample Location	Cable Depth (feet)	Lead (mg/kg)
SED-4A	3.6	3.33
		7.57*
SED-4B		2
SED-4C		1.58

REF-2

Sample Location	Cable Depth (feet)	Lead (mg/kg)
REF2	10.9	0.548

SED-5

Sample Location	Cable Depth (feet)	Lead (mg/kg)
SED-5A	10.7	0.683
		0.811*
SED-5B		0.846
SED-5C		0.816

SED-5-END

Sample Location	Cable Depth (feet)	Lead (mg/kg)
SED-5A-END	8.9	5.57
SED-5B-END		1.77

SED-6

Sample Location	Cable Depth (feet)	Lead (mg/kg)
SED-6A	14.6	0.659
SED-6B		1.18
SED-6C		0.705

Pope Beach

Sample Location	Cable Depth (feet)	Lead (mg/kg)
Pope Beach	1	0.92

Emerald Bay Beach

Sample Location	Cable Depth (feet)	Lead (mg/kg)
Emerald Bay Beach	0.5	1.21

Baldwin Beach

Sample Location	Cable Depth (feet)	Lead (mg/kg)
Baldwin Beach	1	1.33

REF-3

Sample Location	Cable Depth (feet)	Lead (mg/kg)
REF3	8.9	2.45

LEGEND

- Sediment Sampling Location
- Cable Lines

0 1 2 Miles

Notes
1. All samples were collected in July, 2023
2. * = field duplicate sample
3. mg/kg = milligrams per kilogram
4. Samples ending in "A" were collected 15 centimeters (cm) away from the cable.
5. Samples ending in "B" were collected 1 meter (m) away from the cable.
6. Samples ending in "C" were collected 2 m away from the cable.
7. U = not detected above laboratory reporting limits
8. J = estimated concentrations below the laboratory reporting limit and the laboratory detection limit

RAMBOLL 2023 SEDIMENT SAMPLING RESULTS

Lake Tahoe
California

FIGURE 6

RAMBOLL AMERICAS ENGINEERING SOLUTIONS, INC.
A RAMBOLL COMPANY

Table 3. Ramboll Study Results for Sediment Sampling (2023)

Sample ID	Lead (mg/kg)	Cable Depth	Distance From Cable
SED1A-071123-C	**1.28**	8’9”	15 cm
SED1B-071123-C	**1.13**		1 m
SED1C-071123-C	**1.33**		2 m
SED4A-071123-C	**3.33**	3’6”	Directly beneath [a]
SED4A-071123-C-FD*	**7.57**		0-15 cm
SED4B-071123-C	**2.00**		1 m
SED4C-071123-C	**1.58**		2 m
SED5A-071223-C	**0.68**	10.7	15 cm
SED5A-071223-C-FD*	**0.81**		15 cm
SED5B-071223-C	**0.85**		1 m
SED5C-071223-C	**0.82**		2 m
SED5A-END-071223-C	**5.57**	8.9	0 to 10 cm
SED5B-END-071223-C	**1.77**		1 m
SED6A-071223-C	**0.66**	14.6	15 cm
SED6B-071223-C	**1.18**		1 m
SED6C-071223-C	**0.71**		2 m
REF1-071123-R	**0.75**	9.2	Not applicable
REF2-071123-R	**0.55**	10.9	Not applicable
REF3-071123-R	**2.45**	8.9	Not applicable
Meeks Bay-071123-MB	**1.40**	1.6	Not applicable
Emerald Bay Beach EBB-071123-B	**1.21**	0.5	Not applicable
Baldwin Beach-071223-B	**1.33**	1	Not applicable
Pope Beach-071223-B	**0.92**	1	Not applicable

Notes:

[a] Directly beneath the exposed lead core section of the cut cable

mg/kg – milligrams per kilogram
J – reported as an estimated value
* – indicates field duplicate
Bold data indicates a measured value

6.3 Comparison to Background Concentrations and Regulatory Guidelines

The concentrations of lead in Lake Tahoe's surface water and sediment are not elevated above typical background values and are far below any health-based guideline values.

6.3.1 Water Quality of Lake Tahoe Is Not Being Adversely Impacted by the Cables

Lead concentrations in U.S. surface water are variable and, as might be expected, are generally lowest in rural areas. Data collected at five U.S. National Parks, representing natural or "pristine" surface waters, showed median lead levels in surface waters ranging from 0.006 to 0.075 µg/L (ATSDR 2020). Lead concentrations in Lake Tahoe are expected to be similar to these "pristine" areas, and samples collected in Lake Tahoe in 2021 and 2023, near the cables, as well as in reference areas away from the cables, had concentrations that were within this range or were not detectable.

Lake Tahoe results indicate that lead in water samples collected directly above the cables in 2021 by Haley & Aldrich and in 2023 by Ramboll were generally below Haley & Aldrich's method reporting limit (0.3 µg/L) or method detection limit (0.043 µg/L), and below Ramboll's very low method detection limit (0.006 µg/L). Ramboll Station 4 was the only exception to this, but the lead concentrations detected at close proximity to the cable (0.027–0.064 µg/L) were well below federal and state standards for lead in water. Lead results from both the Haley & Aldrich study and the Ramboll study were far below the USEPA's and California Department of Public Health's (DPH) drinking water action level for lead (15 µg/L)[6]; the chronic ambient water quality criterion (AWQC) for aquatic life of 2.5 µg/L; and California's Public Health Goal of 0.2 µg/L (CalEPA 2009; USEPA 2023). Lead concentrations near the cables were also below the average lead concentration estimated for national drinking water supplies across the U.S. (0.9 ug/L–1 µg/L from USEPA (2010) and Bradham et al. 2022, respectively).

Similar to the Haley & Aldrich study, Ramboll's Lake Tahoe field investigation determined that at all locations sampled, the concentrations of lead were similar to background levels reported in Chien et al. (2019), which reports trace metal concentrations and lead isotope ratios in lake water (0.0027 to 0.058 µg/L), river water (0.0021 to 0.14 µg/L), and groundwater (0.013 to 2.4 µg/L) in the Tahoe Basin.[7]

As a result of the findings from both Haley & Aldrich (2021) and Ramboll (2023), it can be concluded that the water quality of Lake Tahoe is not being adversely impacted by the cables.

6.3.2 Sediment Quality of Lake Tahoe Is Not Being Adversely Impacted by the Cables

The Ramboll (2023b) field investigation found that lead concentrations were very low at all measured locations, including cable, reference, and beach stations. Background concentrations of lead in sediment have been established by various sources. According to the National Oceanic and Atmospheric Administration (NOAA) background concentrations of

[6] The action level of 15 µg/L was established in 1991 by USEPA and in 1995 by California.

[7] Lake water, river water, and groundwater concentrations are presented in units of nmol/kg in Chien et al. (2019). Results were converted to µg/L using a conversion factor of 0.2072 µg lead/L water equivalent to 1 nMol lead/kg water.

lead in freshwater sediment are generally expected to fall within the range of 4 to 17 mg/kg (Buchman 2008). All lead measurements in Lake Tahoe sediment were within or below this range.

A study of deep basin cores in Lake Tahoe show that as leaded gasoline use increased beginning in the 1920s, the sediment lead concentrations increased from a baseline of 11–13 mg/kg to more than 80 mg/kg on average, likely due to atmospheric deposition of lead released from cars (Hayvaert 2000). All the nearshore Lake Tahoe samples collected recently were lower than concentrations recorded in deep basin cores.

Because sediment exposures are not common, USEPA and California DTSC do not have human health-based guidelines for lead in sediment. However, they do have health-based guidelines for soil. The California DTSC has a residential soil screening level of 80 mg/kg, which is generally considered acceptable for reuse without restriction, and a commercial/industrial screening level of 500 mg/kg (DTSC 2022). The Lake Tahoe lead concentrations in sediment were below all federal and California-based screening levels and guidelines for lead in soil, including the following:

- DTSC’s soil screening level for residential land use: 80 mg/kg (2022)
- DTSC’s soil screening level for commercial/industrial use: 500 mg/kg (2022)
- USEPA’s regional screening level for lead in residential soil: 200 mg/kg (2024)
- USEPA’s hazard standards for lead in bare soil in play areas: 400 mg/kg (HUD 2012)
- USEPA’s hazard standards for non-play areas of residential yards: 1,200 mg/kg (HUD 2012)

Lake Tahoe lead concentrations in sediment were also well below the mean soil lead concentration of 106 mg/kg for U.S. housing recently reported in the American Healthy Homes Survey II by the U.S. Department of Housing and Urban Development (HUD 2021).

The findings of Ramboll’s sediment study support the conclusion that the lead found in Lake Tahoe sediments in the vicinity of the cables are indistinguishable from background and that the cables do not adversely influence lead concentrations in Lake Tahoe sediment. Furthermore, lead concentrations in Tahoe sediment directly beneath the cables are below-average estimates for residential soil lead based on national datasets and far below health-based guidelines for residential soil.

7. ASSESSMENT OF POTENTIAL LAKE TAHOE HEALTH RISKS

As I described above, there is no evidence that Cables A and B are having a discernable impact on environmental media in Lake Tahoe. Typically, such a situation would not trigger the need for a health risk assessment. Nevertheless, to further evaluate allegations in the complaint I have included a quantitative analysis of potential health risks in my report. I start with the development of an exposure pathway model. The purpose of the exposure pathway model is to determine if there are ways for released chemicals to reach people, flora, and fauna, and if so, what those pathways are.

Lake Tahoe has numerous recreational uses, including beachgoing, swimming, boating, and fishing. It is also a designated source of domestic and municipal drinking water. According to the complaint, members of CSPA use Lake Tahoe to "*fish, boat, kayak, swim, bird watch, view wildlife, and engage in scientific study.*" The complaint also states that CSPA members "*work on and in the waters of Lake Tahoe, making physical contact with those waters on a regular basis*."

The complaint alleges that lead dissolves from the cables and is therefore present in Lake Tahoe water. It then goes on to say that "[*W*]*hen CSPA members contact or drink that water, their body burden of lead is increased and they, and/or their children, face a concomitant increased risk of sterility, neurodevelopmental toxicity, cancer, and other physical ailments associated with exposure to lead*."

To evaluate potential exposure to lead in water, in sediment, and via uptake by fish that are consumed, potential lead concentrations in these media must be measured or estimated. As described in Section 6, the lead concentrations in sediment and surface water collected directly next to the cables are consistent with or lower than national background levels of lead in soil and drinking water. The Lake Tahoe lead concentrations in sediment are also well below available health-based guidelines from USEPA and OEHHA for lead in commercial and residential soil. Surface water concentrations are also well below California and USEPA drinking water standards. As described in Section 7.1.2.1, when lead fish tissue concentrations are modeled using conservative assumptions the fish tissue concentrations are consistent with lead in commercially available fish. Thus, although people potentially can be exposed to lead via Lake Tahoe surface water, sediment, or fish, lead exposures are indistinguishable, and arguably lower, than typical background concentrations.

Potential lead exposures and impacts to blood lead levels associated with exposure to the cables in Lake Tahoe were evaluated using a quantitative exposure assessment and biokinetic models. The methodology and results of this assessment are described in Section 7.1 and my conclusions are present in Section 7.2.

7.1 Quantitative Exposure Assessment

As described in Section 3.1, lead exposure can be assessed by calculating daily lead intakes using exposure assessment principles. These intakes can then be used in biokinetic models to predict blood lead levels in a population. Here, lead intakes in µg/day are estimated using lead concentrations in Lake Tahoe media coupled with exposure parameters from the USEPA Exposure Factor's Handbook, OEHHA, and site-specific information.

Then, the lead intakes are incorporated in biokinetic models to determine whether lead intakes from Lake Tahoe media contribute measurably to geometric mean BLLs in a hypothetical population. The IEUBK model is used to predict BLL contribution for a young child (1 to <6 years of age per the IEUBK model default), and the Adult Lead Model is used to evaluate BLL contribution for adults.[8] Blood lead levels equal to or less than 0.3 µg/dL were considered negligible because they are within the range of normal IEUBK prediction variability, as described in Attachment 2.

The IEUBK and Adult Lead Model inputs and approach for calculating the predicted increase in geometric mean BLL are provided in Attachment 2.

7.1.1 Recreational Exposure

The complaint states that members of CSPA use Lake Tahoe to "*fish, boat, kayak, swim, bird watch, view wildlife, and engage in scientific study.*" Of the recreational activities listed in the complaint, swimming typically results in the greatest potential to contact chemicals in lake water and sediment. While swimming, recreators can incidentally ingest surface water and sediment, resulting in potential exposure to lead. Recreators are also likely to directly contact lake water and sediments via contact with the skin (i.e., dermal contact). However, because dermal absorption of lead is negligible, USEPA does not recommend quantifying the dermal absorption pathway in lead risk assessment (USEPA 1994).[9] Recreators can also indirectly contact constituents in lake water when they consume fish. The fish consumption pathway is discussed in Section 7.1.2.

Lead intake from recreational contact with surface water and sediment was calculated using the following equations and exposure parameters presented in
Tables 4 and 5. The equation for calculating surface water intakes is:

$$Intake_{SW} = C_{SW} \times IR_{SW} \times CF \times ET \times EF$$

Where:

IntakeSW = lead intake from exposure to surface water (µg/day)

Csw = concentration of lead in surface water (µg/L)

IRSW = incidental ingestion rate of surface water while swimming/recreating/working (mL/hr)

CF = conversion factor (1 liter/1,000 milliliters)

ET = exposure time while recreating/working in Lake Tahoe (hr/day)

EF = exposure frequency in days per week (X days/7 days)

The equation for calculating sediment intakes is:

[8] Because California's LeadSpread model is focused on soil exposures, it was not used in this assessment.

[9] OEHHA quantifies dermal exposures in the LeadSpread model; however, the LeadSpread 9 user's guide (OEHHA 2022) notes that dermal absorption has a "negligible effect on estimated blood lead."

$$Intake_{Sed} = C_{Sed} \times RBA \times IR_{Sed} \times CF_A \times ET \times CF_B \times EF$$

$Intake_{Sed}$ = lead intake from exposure to sediment (µg/day)

C_{sed} = concentration of lead in sediment (mg/kg)

RBA = relative bioavailability adjustment for lead in sediment (unitless)

IR_{Sed} = incidental ingestion rate of sediment while swimming/recreating/working (mg/hr)

CF_A = conversion factor (1 kilogram/1,000,000 milligrams)

ET = exposure time while recreating in Lake Tahoe (hr/day)

CF_B = conversion factor (1,000 micrograms [µg]/1 milligram [mg])

EF = exposure frequency in days per week (X days/7 days)

Table 4. Recreational Surface Water Contact Exposure Parameters

Parameter	Scenario /receptor	Surface water exposure near beach		Surface water exposure directly next to severed cable	
		Value	**Source**	**Value**	**Source**
C_{sw} = concentration of lead in surface water (µg/L)	All	0.017 µg/L	Surface water data near beaches, where people are most likely to swim, has not been collected. As a conservative estimate, the average concentration of total lead measured in surface water by Ramboll (2023a) at six sampling stations collected within 6 inches of the cables are used. Depth of cables range from 2 feet 9 inches to 11 feet 10 inches below surface.	0.054 µg/L	Surface water near Station 4 (severed cable) Water depth 2 feet 9 inches Concentration of total lead (average of primary sample and duplicate results) measured in surface water by Ramboll (2023a), collected within 6 inches of severed cable
IR_{SW} = incidental ingestion rate of surface water while swimming/recreating (mL/hr)	Child	96 mL/hour	95th percentile incidental ingestion rate of surface water while swimming for children 6-<11 year of age from Dufour et al. (2006) as recommended by USEPA (2019).	96 mL/hour	95th percentile incidental ingestion rate of surface water while swimming for children 6-<11 year of age from Dufour et al. (2006) as recommended by USEPA (2019).
	Adult	92 mL/hour	95th percentile incidental ingestion rate of surface water while swimming for adults (21+ years of age) from Dufour et al. (2006) as recommended by USEPA (2019).	92 mL/hour	95th percentile incidental ingestion rate of surface water while swimming for adults (21+ years of age) from Dufour et al. (2006) as recommended by USEPA (2019).
ET = exposure time (hr/day)	All	1 hour/day	Site specific information	0.1 hour/day	Site specific information
EF= exposure frequency in days per week (unitless)	All	7/7	Site specific information	1/7	Site specific information

Notes
hr = hour; mg = milligram; kg= kilogram; µg/L =microgram(s) per liter; mL = milliliters; USEPA = United States Environmental Protection Agency

References
Dufour AP, Evans O, Behymer TD, Cantu R. 2006. Water ingestion during swimming activities in a pool: a pilot study. Journal of Water and Health, 4(4), 425-430.
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USEPA. 2019. Update for Chapter 3 of Exposure Factors handbook. EPA/600/R-18/259F. Washington, DC. February.

Table 5. Recreational Sediment Contact Exposure Parameters

Parameter	**Scenario/ receptor**	**Sediment exposure at beach**		**Sediment exposure near cable**	
		Value	**Source**	**Value**	**Source**
C_{sed} = concentration of lead in sediment (mg/kg)	All	1.2 mg/kg	Average of total lead concentrations measured in sediment at Meeks Bay Beach, Emerald Bay Beach, Baldwin Beach, and Pope Beach by Ramboll (2023b).	5.5 mg/kg	Concentration of total lead (average of primary sample and duplicate results) measured in sediment near Station 4A (severed cable) from Ramboll (2023b). Collected from lake bottom, directly beneath exposed lead core of Cable A (1 to 1.5 feet beneath cable, ~4 feet total depth).
RBA = relative bioavailability adjustment for lead in sediment (unitless)	--	0.6	IEUBK/ALM RBA adjustment for lead in soil recommended by USEPA.	0.6	IEUBK/ALM RBA adjustment for lead in soil recommended by USEPA.
IR_{Sed} = incidental ingestion rate of sediment while swimming/recreating (mg/hr)	Child	72 mg/hour	Probabilistic arithmetic mean estimate for incidental ingestion of sediment due to hand-to-mouth contact for children (7 months to 4 years old) from Wilson et al. (2015) as recommended by USEPA (2017).	7.7 mg/hour	Probabilistic arithmetic mean estimate for incidental ingestion of sediment due to surface water intake (all ages).
	Adult	20 mg/hour	Probabilistic arithmetic mean estimate for incidental ingestion of sediment due to hand-to-mouth contact for adults (20-59 years old) from Wilson et al. (2015) as recommended by USEPA (2017).		
ET = exposure time (hr/day)	Recreational	1 hour/day	Site specific information	0.1 hour/day	Site specific information
	Occupational	1 hour/day		0.1 hour/day	
EF= exposure frequency in days per week (unitless)	Recreational	7/7 (1)	Site specific information	1/7	Site specific information
	Occupational	5/7		1/7	

Notes
hr = hour; mg = milligram; kg= kilogram; USEPA = United States Environmental Protection Agency

References
Ramboll. 2023b. Lake Tahoe Sediment Lead Study. August.
USEPA. 2017. Update for Chapter 5 of the Exposure Factors Handbook. EPA/600/R-17/384F. September.
Wilson R, Jones-Otazo H, Petrovic S, Roushorne M, Smith-Munoz L, Williams D, Mitchell I. 2015. Estimation of sediment ingestion rates based on hand-to-mouth contact and incidental surface water ingestion. Human and Ecological Risk Assessment: An International Journal, 21(6), 1700-1713.

The accessibility of the cables varies. In many cases, they are located in water greater than 8 feet deep, so are not easily accessed. In other cases, they are located in shallow water, and in a few locations are visible in sediment on shore.

Two different recreational scenarios were used to evaluate contact with lead in surface water and sediment in the vicinity of the cables. The first scenario assumes typical recreational exposure on a beach. As presented in Section 6.2.3, sediment lead concentrations at four beaches sampled by Ramboll (2023b) were found to be similar, ranging from 0.92 to 1.40 mg/kg (Figure 6 and Table 3). The average sediment lead concentration from the four beaches sampled (1.2 mg/kg) was used for this scenario because they represent popular recreational locations used by swimmers and beachgoers. Surface water concentration data was not collected at recreational beaches, so the average lead concentration measured in surface water within 6 inches of the cables at six sampling stations from Ramboll (2023a) was used as a conservative estimate to approximate lead concentrations in surface water people are exposed to while swimming at the beach.

It was assumed the recreator spends 1 hour/day in contact with surface water and sediment, 7 days per week.

The second scenario assumes a person is swimming directly next to a cable with an exposed lead core. The average of the primary and duplicate results from Station 4 for surface water (Ramboll 2023a) was used to represent the concentration recreators may be exposed to in the unlikely event of swimming directly next to a cable with the exposed lead core. As described in Section 6.2.2, these samples were taken within 6 inches of Cable A where the exposed core is present. It was also assumed that recreators might also incidentally ingest sediment resuspended in the water column that was kicked up while swimming or standing next to the cable, another highly unlikely event. The average of the primary and duplicate results from Station 4A (directly beneath severed cable) from Ramboll (2023b) was used in this analysis. Because this scenario assumes exposure directly adjacent to a severed cable, including at the depth where the cable is present (i.e., several feet underwater), an exposure time of 6 mins, or 0.1 hours, was assumed. It was assumed this exposure happens 1 day/week.

To evaluate exposure to constituents of concern while swimming, the USEPA Exposure Factor's Handbook (2019) recommends upper percentile incidental water ingestion rates of 96 mL/hour for children (6 to <11 years of age) and 92 mL/hour for adults, based on a study by Dufour et al. (2006). An ingestion rate for younger children is not available. The USEPA Exposure Factors Handbook recommends sediment ingestion rates from Wilson et al. (2015) of 72 mg/hour for children (7 months to 4 years old) and 20 mg/day for adults via hand to mouth contact, and 7.7 mg/hour for incidental ingestion of sediment via surface water intake for all ages (USEPA 2017). The hand-to-mouth contact sediment ingestion rates were used for the Baldwin Beach scenario because it is assumed recreators may incidentally ingest sediment on shore while digging, playing with sand etc. The 7.7 mg/hour rate was used to estimate potential lead exposure when swimming directly next to the cable, which accounts for sediment that may be kicked up while swimming and subsequently ingested.

Lead intakes and the associated predicted increases in BLL for surface water and sediment are shown in Table 6 for children and Table 7 for adults. The estimated lead intakes from

surface water and sediment exposure directly next to the exposed cable are so low (<0.001 µg/day) that they cannot be input in the IEUBK model. Thus, potential exposure to lead from swimming directly next to an exposed cable is assumed to result in no measurable increase (<0.3 µg/dL) to BLL. When the sediment and surface water-specific lead intakes for activity at beaches is entered, there is no discernable increase in BLL (i.e., predicted increase in geometric BLL is less than 0.3 µg/dL). When lead intakes for adult recreators are entered in the Adult Lead Model (ALM), there is no measurable increase in geometric mean BLL for any of the media/exposure scenarios evaluated.

Table 6. Modeled Lead Exposure for Children from Swimming

	Lead Intake (µg/day)	Predicted increase in geometric mean blood lead level (µg/dL)
Exposure at beaches		
Surface Water	0.0016	No measurable increase (<0.3 µg/dL)
Sediment	0.052	No measurable increase (<0.3 µg/dL)
Exposure near exposed cable		
Surface Water	0.00007	No measurable increase (<0.3 µg/dL)
Sediment	0.0004	No measurable increase (<0.3 µg/dL)

Table 7. Modeled Lead Exposure for Adults from Swimming

	Lead Intake (µg/day)	Predicted increase in geometric mean blood lead level (µg/dL)
Exposure at beaches		
Surface Water	0.0016	No measurable increase (<0.3 µg/dL)
Sediment	0.014	No measurable increase (<0.3 µg/dL)
Exposure near exposed cable		
Surface Water	0.00007	No measurable increase (<0.3 µg/dL)
Sediment	0.0004	No measurable increase (<0.3 µg/dL)

7.1.2 Fish Consumption

The complaint alleges that humans ingest dissolved lead from the cables when they eat fish caught in Lake Tahoe. In this section, potential lead concentrations are modeled for Lake Tahoe fish using water concentrations measured within six inches of the cables. The modeled Lake Tahoe fish tissue concentration is compared to lead concentrations measured in grocery store fish as reported in the FDA Total Diet Study. Then, the estimated lead intake and impact to BLL potentially associated with Lake Tahoe fish consumption is modeled for children and adults using USEPA's IEUBK and Adult Lead Model, respectively.

7.1.2.1 Estimated Lead Concentrations in Tahoe Fish and Comparison to National Diet Study

Lead uptake in fish tissue is generally minimal, as described in the following paragraph. Nevertheless, lead concentrations in fish tissue can be conservatively estimated according to OEHHA (2012) guidance using a bioaccumulation factor (BAF) and lead concentrations in surface water. Such models are highly uncertain but are used here to determine if further evaluation of this exposure pathway is needed. These modeled lead concentrations are compared to lead concentrations in commercial fish to see if the modeled concentrations for Tahoe fish differ from national background.

As described by OEHHA (2012), lead does not biomagnify in aquatic food chains. Lead chiefly accumulates in the bone, scales, gill, kidney, and liver of fish; it does not accumulate as appreciably in skeletal muscle (i.e., fish fillet). As with any constituent, accumulation of lead in fish typically increases with increasing exposure concentration in water. OEHHA recommends a BAF of 19 for estimating lead uptake in edible (muscle) fish tissue. According to OEHHA (2012), factors that may increase accumulation of cationic metals such as lead in fish include low pH (6.0-6.5 or less) in the water body, low concentrations of aqueous calcium that compete with lead for absorption through the gills, and low concentrations of dissolved organics in water. According to pH data for surface water collected by Ramboll at sampling stations near the cables, pH ranged from 7.01 to 7.94, which includes samples at the surface and near the bottom of the lake. Thus, the lake water is not acidic and is not expected to facilitate increased lead uptake in fish tissue.

The concentration of lead in fish tissue was estimated using the BAF and lead concentration in water using the following equation:

$$C_{Fish} = BAF \times C_{SW}$$

Where:

C_{Fish} = concentration of lead in fish tissue (µg/kg)

BAF = bioaccumulation factor (L/kg)

C_{SW} = concentration of lead in surface water (µg/L)

Conservatively using the mean lead concentration for surface water measured within six inches of the cables (0.017 µg/L) by Ramboll (2023a) and the OEHHA BAF of 19, the estimated lead fish tissue concentration is 0.32 µg/kg. The lead concentration of 0.32 µg/kg estimated for Lake Tahoe fish that potentially uptake lead from water in the vicinity of the cables is consistent with average lead concentrations in commercially available fish sold in grocery stores. Information on concentrations of lead and other chemicals in commercially

available foods comes from the FDA Total Diet Study (FDA 2022b), which analyzes a nationally representative sample of commercially available fish and shellfish commonly consumed in the United States. Of the eight seafoods analyzed in the TDS (fish sticks or patty, canned clam chowder, catfish fillet, cod fillet, salmon fillet, shrimp, tilapia fillet, and canned tuna), lead was detected in fish sticks or patty, canned clam chowder, and shrimp. Of note, the lead reporting limit for these foods was 4 µg/kg, so the conservative estimate of lead in Lake Tahoe fish is less than one-tenth of this limit. Lead was not detected (i.e., lead was less than 4 µg/kg) in the fish fillet samples and canned tuna. Where lead was detected, average lead concentrations were 1.4 µg/kg in fish sticks or patty, 1.7 µg/kg in canned clam chowder, and 4.9 µg/kg in shrimp. These average concentrations include a mix of detected and non-detected results. Of note, the TDS estimates represent lead concentrations after the fish has been cooked, whereas the Lake Tahoe samples are fresh weight (uncooked). Cooked fish samples typically have higher metals concentrations than fresh weight samples because of the moisture that has been lost while cooking. However, even accounting for this difference the Lake Tahoe concentration (0.32 µg/kg) would not increase above 4 µg/kg, meaning the lead level is so low it would fall below the detection limit. This demonstrates lead from cables is unlikely to cause increased fish tissue concentrations, and the modeled tissue concentration estimate is consistent with lead concentrations in fish fillets and other commercially available seafood in the TDS.

7.1.2.2 Estimated Exposure from Fish Consumption

Potential lead intake from fish consumed from Lake Tahoe was estimated using the exposure parameters presented in Table 8. The equation for calculating lead intakes from fish consumption is:

$$Intake_{Fish} = C_{Fish} \times IR_{Fish} \times CF$$

Where:

$Intake_{Fish}$ = lead intake from consumption of fish tissue (µg/day)

C_{Fish} = estimated concentration of lead in fish tissue (µg/kg)

IR_{Fish} = fish consumption rate (g/day)

CF = conversion factor (1 kilogram/1,000 grams)

Table 8. Fish Consumption Exposure Parameters

Parameter	Scenario/ receptor	Value	Source
C_{fish} = concentration of lead in fish tissue (µg/kg)	All	0.32µg/kg	Calculated using approach described in Section 7.1.2.1
IR_{Fish} = fish consumption rate (g/day)	Child	16 g/day	The OEHHA (2008) standard sport fish consumption rate (32 g/day) divided by 2, per OEHHA (2008) recommendation.
	Adult	32 g/day	OEHHA (2008) standard sport fish consumption rate, equivalent to 8 ounces per week.

Notes
ALM = Adult Lead Model; IEUBK = Integrated Exposure Uptake Biokinetic Model for Lead in Children; FAQ = frequently asked questions; OEHHA = Office of Environmental Health Hazard Assessment
g = gram; kg = kilogram; L = liter; µg = microgram
References
OEHHA. 2008. Development of fish contaminant goals and advisory tissue levels (ATLs) for common contaminants in California sport fish: chlordane, DDTS, dieldrin, methylmercury, PCBs, selenium, and toxaphene

As described in Section 7.1.2.1, the fish tissue concentration assumes fish are continuously exposed to water directly next to the cables, i.e., the average of samples taken within 6 inches of the cables by Ramboll (2023a). The standard consumption rate of eight ounces per week (32 g/day) for adults consuming sport fish used by OEHHA (2008) in the development of fish contaminant goals and advisory tissue levels was used in this assessment. This consumption rate was divided in half (16 g/day) when assessing exposure for children, per OEHHA (2008) guidance.

Using these input assumptions, the resultant lead intakes and the associated predicted increases in BLL for fish consumption are shown in Table 9. The estimated increase in geometric mean BLL for children was not measurable (less than 0.3 µg/dL). When the lead intake for adults consuming fish is entered in the ALM, there is also no measurable increase in geometric mean BLL.

Table 9. Lead Exposure from Fish Consumption

	Lead Intake (µg/day)	Predicted increase in geometric mean blood lead level (µg/dL)
Child	0.005	No measurable increase (<0.3 µg/dL)
Adult	0.010	No measurable increase (<0.3 µg/dL)

7.1.3 Occupational Exposure

According to the complaint, members of CSPA *"work on and in the waters of Lake Tahoe, making physical contact with lake waters on a regular basis."* Although not specifically stated

in the complaint, it is assumed workers may also contact sediment. Therefore, the exposure pathways for workers are the same as for adult recreators (incidental ingestion of or dermal contact with surface water and sediment). As stated in Section 7.1.1, dermal absorption of lead is not quantified.

The exposure evaluation for adult recreators provides a conservative estimate of potential lead exposures for workers. The recreational scenario assumes lake water and sediment is ingested while swimming directly next to an exposed cable, or while visiting and swimming at a beach. Most workers are not expected to swim any more frequently than the exposure assumptions used for the recreational scenario (i.e., 1 hour/day, 7 days/week in open water or underwater, directly next to the cable for 6 mins per day, 1 day/week). Thus, the finding of no measurable increase in BLL adult recreators can also be applied to occupational exposures.

Additionally, the lead concentrations measured in sediment by Ramboll (2023b) are far lower than OEHHA's health-based soil screening level of 500 mg/kg for commercial or industrial use. According to the California Department of Toxic Substances Control, this screening level corresponds to the soil lead concentration that would give rise to the 90th percentile estimate of change in blood lead of 1 µg/dL in the fetus of an adult pregnant worker exposed to lead-contaminated soil, calculated using EPA's Adult Lead Model (DTSC 2024). As summarized in Section 6.2.3, the maximum lead concentration measured in sediment by Ramboll (2023b) was 7.57 mg/kg (5.5 mg/kg when primary and duplicate results are averaged), almost two orders of magnitude lower than the commercial industrial screening level. Additionally, contact with sediment near the cables is expected to occur far less frequently than the exposure assumptions on which the OEHHA screening level is based, meaning it is highly protective. Thus, there are no health risks associated with occupational exposure to sediment near the cables.

7.1.4 Drinking Water Exposure

Because drinking water exposure is already included in the IEUBK model, the approach for evaluating lead exposure from Lake Tahoe cable water when used as drinking water differs slightly from the other exposure scenarios. The lead concentration selected to represent Lake Tahoe surface water used as drinking water was used as the drinking water concentration in the IEUBK water menu, and the predicted geometric mean BLL was compared to the IEUBK default. Contributions from lead in drinking water to BLL were not able to be assessed for adults due to limitations in USEPA's Adult Lead Model. However, the IEUBK assessment for children is protective of adults as described in Attachment 2.

The average total lead concentration measured within 6 inches of the cables by Ramboll (0.017 µg/L; 2023a) was used as the drinking water concentration. This assumes the water is not filtered prior to use. Using the average lead concentration from samples taken within 6 inches of the cables is very conservative considering that Tahoe water consumed as surface water is drawn from various parts of the lake, not just directly adjacent to the cables.

The IEUBK default lead drinking water concentration is 0.9 µg/L, while the conservative Lake Tahoe concentration is 0.017 µg/L. The IEUBK estimate is intended to be representative of national average exposures. Based on concentration alone, this suggests consuming Lake Tahoe water as drinking water would decrease lead exposure compared to the national

average. When the surface water lead concentration from Lake Tahoe is used in the IEUBK model, there is no measurable difference in predicted geometric mean BLL (within 0.3 µg/L).

Alternatively, lead intake from surface water consumed as drinking water can be estimated using the exposure parameters detailed in Table 10. The equation to estimate lead intake from drinking water is:

$$Intake_{DW} = C_{SW} \times IR_{DW} \times EF$$

Where:

$Intake_{DW}$ = lead intake from exposure to drinking water (µg/day)

C_{DW} = concentration of lead in drinking water (µg/L)

IR_{DW} = ingestion rate of drinking water (L/day)

EF = exposure frequency in days per week (X days/7 days)

Table 10. Drinking Water Exposure Parameters

Parameter	Scenario/ receptor	U.S. Average Drinking Water Intake		Drinking (Surface) Water near Cables	
		Value	**Source**	**Value**	**Source**
C_{sw} = concentration of lead in drinking water (µg/L)	All	0.9 µg/L	IEUBK v2 default	0.017 µg/L	Average concentration of total lead measured in surface water by Ramboll (2023a), collected within 6 inches of cables.
IR_{DW} = ingestion rate of drinking (L/day)	Child	0.53 L/day	Age-weighted drinking water rate for children 1 to <6 years from USEPA's IEUBK model v2.0	0.53 L/day	Age-weighted drinking water rate for children 1 to <6 years from USEPA's IEUBK model v2.0
	Adult	1.28 L/day	Mean consumers-only drinking water ingestion rate for adults 21 to <50 years from USEPA Exposure Factors Handbook (USEPA 2019)	1.28 L/day	Mean consumers-only drinking water ingestion rate for adults 21 to <50 years from USEPA Exposure Factors Handbook (USEPA 2019)

Notes
hr = hour; mg = milligram; kg= kilogram; µg/L =microgram(s) per liter; mL = milliliters; USEPA = United States Environmental Protection Agency

References
Ramboll. 2023a. Lake Tahoe Water Lead Study. July.
USEPA. 2019. Update for Chapter 3 of Exposure Factors handbook. EPA/600/R-18/259F. Washington, DC. February.

The lead intakes and predicted increases in geometric mean BLL associated with drinking water (Lake Tahoe surface water and national) are shown in Table 11. Both the intakes and impacts to BLL are below the OEHHA thresholds. Additionally, the lead intakes are much greater for the national drinking water dataset than the Lake Tahoe surface water dataset.

Table 11. Lead Exposure from Drinking Water

	Lead Intake (µg/day)	**Predicted increase in geometric mean blood lead level (µg/dL)**
Child		
Tahoe	0.009	Within 0.3 µg/dL [a]
National Drinking Water	0.5	<0.3 µg/dL
Adult		
Tahoe	0.022	Not calculated [b]
National Drinking Water	1.2	Not calculated [b]

Notes
[a] Using the Tahoe water concentration, the geometric mean blood lead level predicted by IEUBK is less than the geometric mean blood lead level predicted by IEUBK when the IEUBK default drinking water concentration is used. But the difference in predicted geometric means is within the range of model error (0.3 µg/dL)
[b] Could not be calculated because the Adult Lead Model cannot evaluate drinking water exposures. See Attachment 2.

7.2 Consideration of Potential Lake Tahoe Exposures Confirms That There Is No Health Risk

The quantitative risk analysis presented in Section 7.1 confirms that, contrary to the allegations in the CSPA complaint, lead from Cable A and Cable B does not pose a health risk to people who use Lake Tahoe to “fish, boat, kayak, swim, bird watch, view wildlife, and engage in scientific study.” Nor is there any health risk for those who “work on and in the waters of Lake Tahoe, making physical contact with those waters on a regular basis.” Risks were quantified for swimming scenarios, fish consumption, and drinking the water using assumptions that are likely to have greatly overstated the potential exposures. Even with these conservative assumptions, lead intakes are predicted to be negligible. The predicted intakes also did not produce any measurable increases in predicted BLLs. The scenarios selected for quantification are also protective of other activities listed in the complaint. As described, the swimming scenario will be protective for those working in and around the water. This scenario is also protective for those who boat, kayak, or fish in the lake and for those who birdwatch and view wildlife near or on the lake.

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ATTACHMENT 1
CURRICULUM VITAE OF
DR. ROSALIND SCHOOF

RAMBOLL

ENVIRONMENT & HEALTH

ROSALIND A. SCHOOF

PhD, DABT, Fellow ATS
Principal

Dr. Rosalind Schoof is a board certified toxicologist with more than 35 years' experience assessing human health effects and exposures from chemical substances in a variety of settings, such as contaminated sites, commercial/ industrial/agricultural/residential projects, product uses, dietary exposures and general home and community exposures. Her projects have included numerous formal health risk assessments conducted under various US and international regulatory settings, as well as regulatory, research and litigation projects. Dr. Schoof has directed evaluations of chemical toxicity, derivation of risk-based exposure levels, health risk assessments for cancer and noncancer end points and multimedia exposure assessments.

Dr. Schoof is an internationally recognized expert on evaluation of arsenic and metals in the environment and in the diet, and on the bioavailability of metals from soil with over 35 peer-reviewed publications. She has served on numerous peer review panels for US agencies and Canadian ministries, on several National Research Council committees, and the US Department of Defense Strategic Environmental Research and Development Program (SERDP) Science Advisory Board. Prior to her consulting career, Dr. Schoof worked for a pharmaceutical company conducting safety assessments for new drugs, and designing and directing toxicity studies. She also worked in the Office of Toxic Substances at USEPA.

EDUCATION

1982
PhD, Toxicology
University of Cincinnati, Cincinnati, OH, United States

1975
BA, Molecular Biology
Wellesley College, Wellesley, MA, United States

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CREDENTIALS

Registrations and Certifications

- Diplomate, American Board of Toxicology (certified in 1986; recertified in 1991, 1996, 2001, 2006, 2011, 2016, and 2021)
- Fellow, Academy of Toxicological Sciences

Professional Affiliations and Activities

- Society of Toxicology
- International Society for Exposure Science

PROJECTS

Exposure Studies and Risk Assessment for Mine and Smelter Sites

- Probabilistic Risk Assessments at mine/smelter sites, Arizona (2020-present). Directed probabilistic risk assessment for arsenic exposure at multiple sites in Arizona potentially affected by mine tailings or smelter emissions.
- Lead Bioavailability Soil Amendment Study (2017-present). Directed a study of the effectiveness of multiple soil amendments in reducing the bioavailability of soil lead. The study was a collaborative research project among a mining company, EPA and Tribal scientists with a laboratory phase at a university and a field study at a site in western Washington State.
- Risk Assessment for Copper Refinery Site, Montana USA (2014-present). Oversaw a work plan and multipathway human health risk assessment for a community adjacent to a former copper refinery. Lead and arsenic were the constituents of potential concern investigated in the risk assessment. Exposures via contact with soil and house dust were calculated based on soil and dust samples collected from residential and commercial properties. Site-specific soil bioavailability data was applied in estimating exposures, and soil contact was not assumed to occur during the portion of the year when ground was frozen and snow-covered. Calculation of preliminary remediation goals for residential and commercial properties was performed. Ongoing work addresses additional operable units.
- Coal Mining Water Quality Assessment in British Columbia (2014-2021). Provided human health risk assessment support for the Elk Valley Water Quality Plan (EVWQP) conducted under an order from the British Columbia Ministry of Environment. The EVWQP is an area-based management plan to address current water quality trends for selenium, cadmium, nitrate, and sulphate and calcite formation. Two successive health risk evaluations were prepared with oversight from advisory groups including Provincial, Federal and First Nations representatives.
- Evaluation of Historical Mining Activity and Blood Lead Levels, Montana USA (2012-present). For more than a decade, blood lead samples have been collected and analyzed by public health programs in a Montana community where mining and mineral processing activities have occurred for more than a century. For the Phase 1 study, an electronic database was compiled from health department paper records that includes over 6600 records for infants, children and pregnant women. The study focused on over 3,500 records for Butte children 12 to 60 months old for the period from 2003 through 2010. A reference population was created by considering the comparability of prominent risk factors between the Butte dataset and a national blood lead database. Comparisons with the reference population and assessment of variations in blood lead levels across neighborhoods guided the assessment of ongoing remediation and lead abatement activities. Study results were published in a peer-reviewed journal. A Phase 2 study extended the analysis of blood lead data through 2017, and planning has begun for a phase 3 study.
- Evaluation of Smelter Wastes in a River and Reservoir, Washington USA (2006-present). Assisted with planning for site investigation, exposure study and human health risk assessment of slag and other smelter wastes present in a large reservoir. Participated in coordination with two tribes and state and federal agencies. Continue to provide input to ongoing site investigations and risk analyses.
- Voluntary Cleanup of Mining Site, Colorado USA (2020-present). Provided analyses of potential lead exposures to inform development of soil action levels, interacted with regulators and participated in public meetings.
- Biomonitoring Study in Former Smelter Community, Montana USA (2012-2014). Directed a blood lead and urine arsenic biomonitoring study at a former copper smelter site in Montana. The study included more than 100 participants who filled out a questionnaire designed to collect relevant demographic information and to identify additional sources of lead or arsenic exposure. Individual results were provided to participants, and a report on community-wide results was prepared.

- Arsenic Inhalation/Smelter Risk Assessment, Namibia (2016). Evaluated health risks for inhalation of airborne arsenic by communities living in the vicinity of an operating copper smelter. Applied unique approaches for consideration of formal and informal residential areas. Due to lack of country-specific guidance, utilized Canadian, South African, and other international guidelines.
- Urine Arsenic Biomonitoring Before and After Remediation in a Mining Community, Arizona USA (2011-2012). Directed a urine arsenic biomonitoring study in a small mining community in Arizona. The study was conducted in two phases, before and after completion of residential yard soil removal activities. Results were provided confidentially to individual participants and community-wide results were shared at a public meeting.
- Risk Assessments for Deloro Mine Site, Ontario (2010-2011). On behalf of the Ontario Ministry of Environment, directed human health risk assessment updates for onsite and offsite exposures to arsenic and metals. Primary populations of concern included on-site workers and trespassers and off-site recreational visitors. Calculation of preliminary remediation goals was performed for both on- and off-site portions of the study area.
- Evaluation of Risks at Former Uranium Mine, Alaska USA (2009-2011). Assisted with planning for site investigation and human health risk assessment of metals, radionuclides and radon from waste rock. Participated in meetings with state, federal and Native community representatives.
- Evaluation of Metal Risks from an Operating Lead and Zinc Smelter, British Columbia (1990s-2014). Multiple risk assessment projects. Analysis of metal uptake into home-grown produce. Oversaw multipathway probabilistic risk assessment for exposures to arsenic, cadmium and other metals in a community with an operating lead and zinc smelter. Conducted earlier phases of the risk assessment on behalf of a community stakeholder group. Presented plans and results to stakeholders and government representatives.
- Metal Smelter Risk Assessment, South America (2005-2008). Directed assessment of current and future risks from sulfur dioxide, particulates, lead and other metals from an operating smelter in support of an extension of an operating agreement. The study included collection of outdoor dust, indoor dust, soil and drinking water. Air modeling and a diet study were conducted by collaborators. Exposures of children to lead were assessed using a probabilistic exposure model that was modified to predict recently collected blood lead data. The adult lead model was also modified to better reflect site conditions. Presented findings at public meetings attended by thousands of residents and workers. Update conducted after three years confirmed model predictions.
- Former Copper Mining Site Risk Assessment, Nevada USA (2002-2020). Multiple risk assessment projects. Directed study plans of risk assessments for potential exposures to metals and radionuclides from tailings and other wastes at a former copper mining site with multiple operable units being evaluated under CERCLA. Participated in coordination with tribes and other local stakeholders.
- Former Uranium Mine Cleanup Negotiations, Washington USA (2005). Provided comments on validity of a baseline human health risk assessment and rationale for cleanup plans for metals and radionuclides at a former uranium.
- Risk Assessment for Voluntary Cleanup of Mining Site, Colorado USA (2005). Directed an assessment of risks from residential and recreational exposures to lead for a community affected by historic mining activities. The risk assessment was part of a site investigation under the 1994 Colorado Voluntary Cleanup and Redevelopment Act. Directed studies of the relative bioavailability and mineralogy of lead in site soils. Presented plans at community meetings and participated in negotiations with state agency staff.

- Blood Lead Exposure Study for Mining Site, Colorado USA (2006-2007). Conducted a comprehensive community-wide blood lead and environmental monitoring study. The study was conducted in two phases. The study objectives were to further characterize current blood lead levels and identify factors influencing exposures, and understand seasonal fluctuations in blood lead levels in order to characterize the potential contribution of the soil contact exposure pathway to blood lead levels. High participation rates (67 percent and 63 percent of all eligible households for Phases I and II, respectively) and the high rate of re-participation (82 percent) for the Phase II sampling event ensured that the measured levels of lead in blood and environmental media were representative of exposures to residents. Yard soil data had been previously collected from all yards. During the blood lead study, house dust and drinking water samples were collected and paint lead measurements were taken. A questionnaire was administered to gather demographic information and to identify additional possible sources of exposure.
- Metal Processing Site Risk and Bioavailability Support, Utah USA (2002-2004). Provided human health risk assessment support for evaluation of lead and arsenic in soils at a former mineral processing site in Utah. The site is currently a wildlife refuge. Recreational exposures onsite and exposures in an adjacent residential area were evaluated. *In vitro* bioavailability studies and mineralogical evaluations of lead and arsenic were conducted, and results used in the site risk assessment.
- Mining Site Risk Assessment California USA (2002). Conducted a preliminary human health risk assessment evaluating metals in sediments, surface water, and other media downstream of a copper and sulfur mine site in California. Exposure scenarios included both recreational and Native American activities.
- Historic Copper Mine Site, Montana USA. Evaluated potential exposures and risks posed by elemental mercury, lead and arsenic in basements of homes built on a former historic copper mine site in Montana. Potential exposures to lead, arsenic and mercury in attic dust, indoor dust in living areas and in soils was also evaluated.
- Former Copper Smelter Site, Washington USA. Assembled and summarized literature related to assessment of exposures to arsenic in soil for negotiations and legal action related to a former smelter site in Washington State. Attended stakeholder meetings and made presentations on arsenic toxicity and risk assessment. Reviewed and commented on state documents. Prepared affidavit addressing issues related to arsenic toxicity and risk assessment.
- Zinc Smelter Risk Assessment and Bioavailability Research Program, Oklahoma USA (1994). Managed human health risk assessment tasks for the work plan, remedial investigation and feasibility study of cadmium, lead and arsenic in soil at a former zinc smelter site in Bartlesville, Oklahoma. Planned for collection of site-specific data to fill gaps in USEPA's baseline human health risk assessment, including paired soil and indoor dust samples, "hot spot" delineation, and a bioavailability study of cadmium and lead in soil. Directed development of revised remediation goals for arsenic, cadmium and lead using site specific data and wrote position papers supporting the recommended goals. Consideration of reduced bioavailability from soil and reduced toxicity in the presence of zinc resulted in soil cadmium cleanup levels of 100 and 200 mg/kg for residential and occupational land use, respectively. A lead cleanup level for occupational areas was derived using an adult lead exposure model. Monte Carlo analyses were used to document protectiveness of cadmium and arsenic cleanup levels. Presented plans and results to USEPA and state staff, and at public meetings. Assisted in negotiating cleanup levels for cadmium, lead and arsenic that subsequently reduced remediation scope and cost by $50 million.

- Mining and Smelting Site Strategic Risk Assessment Support USA (1990-1998). Over an eight-year period, managed a multi-site general risk assessment support contract for mining and smelting sites throughout the Rocky Mountain region. In addition to providing risk assessment support for specific sites, responsibilities included ensuring that risk assessment strategies and positions were consistent from site to site and that risk assessment strategies were coordinated with litigation strategies. Identified pivotal sources of uncertainty affecting risk estimates for many sites, and helped design and conduct research to support more realistic assessments of risks. Presentations were regularly made to USEPA and state staff on behalf of the client.
- Copper Mine Site Risk Assessment Program, Montana USA. Managed preparation of a series of position papers describing the proper methods for evaluating exposures to lead and arsenic from mining wastes in soils, groundwater and surface water in Butte, Montana. Soil issues included evaluation of the uncertainties associated with USEPA's oral carcinogenicity assessment for arsenic, bioavailability of lead and arsenic in soils, and discussion of appropriate ways to apply the uptake biokinetic model and community blood-lead studies to assessments of lead exposures. Prepared documents describing the proper methods to evaluate risks from groundwater and surface water contaminated with arsenic, lead, cadmium and other metals released as mining by-products at several operable units. Comments were also prepared on baseline risk assessments and preliminary remedial goals from USEPA and state agencies.
- Reservoir Sediment Risk Evaluation, Montana USA. Assisted in preparing a document describing the proper methods of evaluating human health risks associated with recreational exposures to arsenic, cadmium and lead in sediments at a reservoir in Montana.
- Lead Mining District Risk Evaluations, Colorado USA. Advised client of best methods for assessing lead exposures at a historic mining site in the Rocky Mountains. Described available data and appropriate methods for comparing the bioavailability of lead from soil, slag, mining wastes and tailings. Critiques were provided for community blood lead studies and the application of the uptake biokinetic model to assess lead exposures at the site.
- Risk-Based Cleanup Goals for a Barium Ore Site, California USA. Provided strategic guidance and senior review for development of risk-based preliminary remediation goals for barium in soil at a former ore processing plant in Modesto, California, that was expected to be redeveloped as an industrial or recreational park.
- Risk Assessment for Voluntary Cleanup of Mining Site, Colorado USA. Directed the development of risk assessment strategy for a mining site being addressed by the client under the 1994 Colorado Voluntary Cleanup and Redevelopment Act. Chemicals evaluated included arsenic, lead and manganese. Extensive background investigations were conducted.
- Lead Smelter Risk Evaluations, Utah USA. Provided risk assessment support for agency negotiations, site investigation strategy and document review for several smelter sites in the Salt Lake Valley. Evaluated risks associated with arsenic, cadmium and lead in soil and slag, considering residential and occupational exposures. Provided comments on USEPA risk assessment and represented the client in meetings with the agency. Designed and directed a study of lead bioavailability in rats, in which site soils containing lead were added to the rat diet.

Risk Assessment of Manufacturing, Landfill and Waste Combustion Sites

- Soil Arsenic Bioavailability (2022-2023). Advised a client on soil arsenic bioaccessibility testing and calculation of action levels.

- Pesticide Manufacturing Facility Evaluation, New York USA (1995-2020). Directed health risk assessments and development of risk-based cleanup levels for arsenic in offsite soils for multiple operable units near a former arsenical pesticide manufacturing facility in New York. Offsite areas included a public school complex. Made multiple presentations of client's position to the New York State Department of Environmental Conservation RCRA staff and the New York State Department of Health staff. Made presentations to public advisory groups for the facility and at school board and community meetings.
- Pesticide Testing Facility, Florida USA (2010-2015). Directed development of a system for prioritizing chemicals for sampling at pesticide testing facility used to test a variety of pesticides used for agricultural, horticultural, and silvacultural purposes. Scientific information about the environmental and toxicological properties of these chemicals was obtained to support development of a comprehensive remediation plan for the site. Using chemical property and toxicity information, pared a list of more than 400 chemicals, including unnamed test products, to approximately 30 for a targeted soil investigation.
- Prudhoe Bay RCRA Site Investigation, Alaska USA (2012). Provided peer review of human health risk assessment documents and other documents related to investigation of several subunits within the Prudhoe Bay RCRA site. Documents included several conceptual site reports and the human health and ecological risk assessment for unexcavated inactive production reserve pits, a barium bioavailability soil sampling report, a consolidated background report, and a preliminary review of the interim measures system evaluation including the hydrologic conditions at the site (specifically those hydrologic conditions which may impact fate and transport and the chemical source characterization), as well as the preliminary review and evaluation of the adequacy of a remedial system installed at the site in the 1980s.
- Soil Mercury Exposure Analyses. Supported analyses of role of soil mercury speciation and bioavailability in moderating potential mercury exposures near a former battery manufacturing site in New York.
- PAH Risks at Former Coal Gasification Site. Provided human health risk support to a city parks department evaluating risks from residual PAHs in soil.
- Risk Evaluation for West Virginia Brownfield (Landfill) Site. Directed evaluations of human health and ecological risks associated with sediment and soil affected by releases from an historic landfill along the Kanawha River in Nitro, West Virginia. Risk assessments were used to support a voluntary cleanup application so that the site could be redeveloped as a boat ramp park. Presented findings at city council meetings and a meeting of the citizen's advisory group.
- Brownfield Human Health Risk Assessment, South Charleston, West Virginia. Performed evaluations of risks associated with soils, groundwater and a river affected by chlorinated organic compounds released from a former carbon tetrachloride production facility. The risk evaluations were conducted in accordance with the tiered system established by the state for the voluntary remediation program. Focus of the evaluation was on assessing potential risks from exposures to volatile chlorinated organic compounds that were infiltrating a planned new commercial buildings as parcels are put up for sale and utility and construction trenches. USEPA's latest versions (2001) of the Johnson & Ettinger advanced building infiltration via groundwater and soil models were used to evaluate potential impacts to indoor air in future buildings. Participated in stakeholder meetings and public briefings regarding assessments of potential site risks before and after redevelopment.
- Human Health Risk Assessment for a Wood Treating Facility, Minnesota. Oversaw an extensive human health risk assessment for a confidential wood treating facility in Minnesota that included traditional tribal lifeways of Native Americans living in the community. Chemicals of potential concern included PCBs, carcinogenic PAHs, pentachlorophenol and dioxins/furans.

- Brownfield Redevelopment Risks. Provided human health risk support to a midwestern city environmental department for several redevelopment properties with possible arsenic and lead contamination. Activities included evaluation of background concentrations and human health risk assessment.
- Chemical Distribution Facility Risk Communication, New Jersey. Prepared fact sheets for a chemical distribution facility with chlorinated volatile organic chemicals present in soil and groundwater. Fact sheets were distributed to neighboring businesses as part of an indemnity and access agreement. Preparation of fact sheets required reviewing site data, evaluating vapor intrusion modeling, identifying chemicals of potential concern, researching chemical toxicity, and determining the nature of potential exposures and likelihood of these exposures being of concern.
- Brownfield Redevelopment Vapor Intrusion Risk Assessment, California. For large industrial/commercial Brownfield redevelopment project in California, Conducted an indoor air risk assessment using the Johnson & Ettinger model to assess risks from chlorinated solvents in soil and groundwater. Presented findings to potential purchaser and to state regulators.
- Hazardous Waste Combustor Risk Assessment Work Plan, Idaho. Managed preparation of human health risk assessment work plans for a planned hazardous waste combustor at a phosphorous production facility in Idaho. Project included extensive negotiations with USEPA and tribal representatives, as well as intensive coordination with the engineering design team.
- Evaluation of Nickel Carcinogenicity. Critically evaluated the potential carcinogenicity of different forms of nickel present at a former steel mill site.
- Mercury Bioavailability Research and Risk Analysis, New Jersey. Provided guidance for risk assessment strategy at a former manufacturing site in New Jersey with mercury and dioxin contamination. Guided the design of studies of mercury bioavailability from soil, and prepared a comprehensive report presenting the results and justifying the selected approach for submittal to the state. Directed study of evaluating mercury vapor release from soil. Assisted in a comparison of dioxin data with regional background values.
- Assessment of Soil Arsenic Background and Risks, Illinois. Provided support in assessing potential risks associated with arsenic in the soil in residential areas surrounding an operating glass factory in Illinois. Prepared a presentation for the Illinois EPA and Department of Health, and developed statistically defensible sampling plans to compare site concentrations to background. Prepared a comprehensive report proposing an innovative approach to identifying safe arsenic concentrations in soil for submittal to the state.
- Pesticide Manufacturing Facility, Texas. Prepared an exposure pathway analysis for a former pesticide manufacturing facility in Texas with elevated concentrations of arsenic in soil, interior dust, groundwater, surface water and lake sediments. Assisted in designing a soil sampling plan for site investigations overseen by the Texas Natural Resources Conservation Commission.
- Evaluation of Vapor Intrusion from Groundwater, Minnesota. Provided support in assessing potential exposures of residents and workers at a site in Minnesota to chlorinated volatile hydrocarbons in a groundwater plume. The chemicals evaluated include vinyl chloride, 1-dichloroethene, trichloroethene and tetrachloroethene. Exposure pathways include infiltration from groundwater into basement air.
- Risk Evaluation of Chromium in Groundwater, Montana. Directed a screening-level evaluation of human health and ecological risks associated with chromium (VI) in a golf course pond and ditch in Montana. Evaluated transport and fate of chromium in a groundwater plume, and potential impacts to a nearby river. Efforts supported a response action based on natural attenuation, with groundwater monitoring and a continuation of existing institutional controls on groundwater and land use.

- Navy Site Risk Assessment Support. Provided strategic guidance for risk assessment efforts for several Navy facilities. Participated in stakeholder meetings and reviewed draft documents.
- Wood Treatment Site Risk Evaluations, Butte, Montana. Managed preparation of documents describing the proper methods of evaluating human health and ecological risks from PCP, PAH and dioxin contamination at a pole-treating plant in Butte, Montana. Assessed risks from exposures to soil, groundwater, surface water and air.
- Bulk Fuel Terminal Risk Evaluation, Seattle, Washington. Provided senior review for a project to develop risk-based cleanup levels for a former bulk fuel terminal in Seattle, Washington.
- Bulk Fuel Facility Risk Evaluation, Kirkland, Washington. Provided senior review for the development of soil and groundwater cleanup levels for a former bulk fuel facility, currently being used as a municipal park, in Kirkland, Washington. Cleanup standards were developed using Washington State's Model Toxics Control Act Method B. Substances included petroleum constituents such as BTEX and PAH compounds.
- Gas Station Risk Evaluation, Seattle, Washington. Provided senior review for development of soil cleanup levels, using Washington State's Model Toxics Control Act Method B, for a former gas station in Seattle, Washington.
- Risk Evaluation for Petroleum Transfer Station, Alaska. Assisted in preparing a work plan and risk assessment that included development of cleanup levels for benzene-contaminated groundwater from leakage of petroleum products at a transfer station in Alaska. Groundwater was demonstrated to be unsuitable as a domestic water source, and the need for remediation was based on potential exposures to volatile chemicals transferred into homes from the basement or ground level.
- Navy Facility Risk Evaluations, Washington. Served as project manager for USEPA technical enforcement support oversight activities at several US Navy NPL sites in Washington State. Provided human health risk assessment guidance, and coordinated review of all aspects of remedial investigation work plans and reports. Contaminants included chlorinated hydrocarbons, solvents, metals and fuels. Primary exposure pathways included groundwater, surface water and marine organisms exposed to contaminated sediments.
- Municipal Incinerator Risk Assessment, Seattle, Washington. For the City of Seattle Solid Waste Utility, performed public health and risk analysis for a municipal incinerator as part of an EIS on waste reduction, recycling and disposal alternatives. Assessed risks from stack emissions of metals, dioxins and other organic compounds. Presented methods and results to local, state, and federal officials, environmental groups, the public and a peer review committee.
- Hazardous Waste Incinerator Risk Evaluation, Florida. Directed human health and ecological risk assessment support activities for a private client opposing the permit application for a hazardous waste incinerator in Polk County, Florida. Critiqued a risk assessment submitted to the state in support of the permit for the incinerator.
- Hazardous Waste Incinerator Risk Evaluation, New Jersey. Performed a preliminary risk assessment for the development of a hazardous waste incinerator in New Jersey.
- Hazardous Waste Incinerator Risk Assessment Peer Evaluation, Kentucky. Provided extensive peer review comments on methods and results of a risk assessment on a hazardous waste incinerator in Kentucky.
- Municipal Incinerator Risk Analyses, Washington. Developed procedures and preliminary assessments for a municipal incinerator planned by a Native American tribe in Washington State.
- Petroleum Refinery RCRA Risk Assessment, Colorado. Prepared a human and environmental assessment work plan for a RCRA facility investigation of a petroleum refinery. Key contaminants included BTEX, PAHs and chlorinated hydrocarbons.

- Underground Storage Tank Evaluation, Alaska. For the Alaska Department of Environmental Conservation (as subcontractor, determined technical requirements and critically reviewed risk assessment and proposed groundwater cleanup levels for a gasoline leak from an underground storage tank. Provided guidance for risk management strategy.
- Hazardous Waste Site Risk Assessments, Oregon. For the Oregon Department of Environmental Quality, provided strategic guidance and senior review for two risk assessments on a hazardous waste site, including a baseline risk assessment conducted for a former wood-treatment facility that used PCP, creosote, and arsenical fungicides and a screening-level human health risk assessment for a hazardous waste site located in a unique desert environment. Key issues evaluated at the wood-treating facility included uncertainties in the slope factor for PCDDs and PCDFs and the comparative risks associated with consumption of fish and crayfish from reference locations. Potential contaminants of concern at the desert site included PCDDs and PCDFs, chlorinated phenoxy herbicides, lead, TCE and benzene.
- Chloralkali Manufacturing Site Risks, New York. Provided senior review and guidance for an assessment of risks associated with mercury and PCBs in fish in Onondaga Lake. The impact of a former chloralkali facility on site risks was evaluated in comparison to the impacts of other sources in Onondaga and other comparable lakes.
- Munitions Facility Risk Evaluations, Oregon. Provided human health risk assessment guidance and work plan review for CERCLA and RCRA investigations of a federal facility in Oregon contaminated with munitions.
- Bioavailability of Metals in Refinery Soil, New Jersey. Provided strategic guidance and senior review for an evaluation of the bioavailability of arsenic, beryllium and lead from soil at an operating refinery in New Jersey.
- Manufacturing Facility Risk Assessment, Ohio. Provided senior review for human health risk assessment components of an expedited RI/FS for an alloy and chemical production facility in Ohio that has produced both radiological and chemical wastes. Tasks include designing and implementing the baseline risk assessment for an operating facility, and participating in the selection of cleanup levels and remedial actions. Primary chemicals of concern include arsenic, chromium, vanadium and the radionuclide decay chains of thorium-232 and uranium-238.
- Pulp Mill Risk Assessment, Alaska. Provided senior review of human health risk assessment issues for an RI/FS of marine areas potentially affected by releases from a pulp mill. Reviewed a work plan to identify potential human health risks associated with exposure to substances in sediments that may bioaccumulate to fish. Key issues include identifying appropriate background concentrations of PCDDs/PCDFs in fish and shellfish in the region and at other US locations and selecting representative fish consumption rates for use in the risk assessment.
- Assessment of Mercury Risks for Instrument Manufacturing Site, Rochester, New York. Provided senior review for the development of alternative cleanup levels for mercury in site soils using site-specific bioavailability data from a former instrument manufacturing facility in Rochester, New York.
- Manufacturing Facility Risk Assessment, California. Provided technical review for a comprehensive baseline human health risk assessment for a former manufacturing facility in southern California. More than 30 chemicals of potential health concern were detected in soil, groundwater, or ambient air, including BTEX, nitro, phenolic and chlorinated organic compounds.
- Military Installation Risk Assessment, San Francisco, California. Provided strategic guidance and senior review for multipathway human health and ecological risk assessments for a military installation, which comprised 11 major study areas and more than 40 individual sites. The risk assessments were used to support the selection of sites to be considered in the feasibility study and for the development of preliminary soil cleanup levels. Chemicals of concern included metals, volatile organic compounds, PAHs, PCBs and pesticides.

- Appliance Manufacturing Site Risk Assessment, Ohio. Assisted in the development of an approach used to assess human health risks from lead in soil and sediment at a television manufacturing facility.

Community and Product Risk and Exposure Assessments

- Lead in Water Fountains (2022-2023). Advised a company about testing and interpretation or results of tests for lead in drinking water fountains in a manufacturing facility.
- Assessment of Plastic Aquaculture Gear (2021-present). Provided a technical memorandum and updates to a summary of studies of microplastics in seafood and the sources, potential addition of microplastics to the marine environment via degradation and fragmentation of plastic farm gear, and potential leaching of harmful substances from aquaculture gear and microplastics.
- Assessment of Polyfluoroalkyl Substances (PFAS) in Plumbing Products (2020-2022). Advised on testing of potential for PFAS leaching from fixtures.
- Assessment of Polyfluoroalkyl Substances (PFAS) in Ski Wax (2020). Supported response to USEPA Toxic Substances Control Act action related to imported ski waxes alleged to contain PFAS compounds.
- Lead and Copper in Hotel Drinking Water (2019-2020). Evaluated if the sampling methodology used to test drinking water sources at a hotel in Canada were consistent with the latest version of the Guidelines for Canadian Drinking Water Quality (GCDWQ) for lead and copper, and if the results were below the health-based guideline for both parameters and the aesthetic guideline for copper.
- Lead Paint Release from TV Towers (2018). Directed an investigation of potential impacts of lead paint release from cleaning of TV towers in a residential neighborhood under EPA emergency response.
- Assessment of Mercury in Soils (2017-2018). Evaluated potential human health risks posed by naturally-occurring mercury at a residential development in northern California. Mercury speciation and relative bioavailability were assessed. Potential risks to on-site residents and construction/maintenance workers were assessed using both default and site-specific exposure parameters taking into account site-specific conditions and exposure pathways.
- Assessment of Cadmium in Oysters (2017). Prepared a response to a Proposition 65 claim that cadmium concentrations in smoked oysters should trigger a "Prop 65" warning label. Cadmium was naturally present in the oysters. Claim was settled favourably.
- Assessment of Plastic Aquaculture Gear (2017). The Monterey Bay Aquarium Seafood Watch Program issued an updated evaluation of farmed pacific geoduck in December 2016. Prepared an analysis showing that many of the concerns raised regarding plastic debris and microplastics are not likely to be associated with geoduck aquaculture gear and practices.
- Copper in Drinking Water (2016). Provided advice to condominium association in southern California regarding copper in drinking water.
- Arsenic Trioxide Product Stewardship and Safety Analysis (2015). Preliminary evaluation of regulatory, safety and public perception issues associated with sale of arsenic trioxide for use in pesticides and wood preservatives.
- Assessment of Arsenic in Geoduck Clams (2013-2014). Assisted Washington State shellfish growers in responding to a Chinese ban on imports of geoducks asserted to have elevated arsenic concentrations.
- Risks of PAH in Coal Tar Products (2010). Provided critical review of study of influence of coal tar sealcoat on house dust PAH concentrations.

- Asbestos Exposure and Risk Evaluation for Sediment/Soil, Swift Creek WA. Conducted an analysis on behalf of the county in support of wetland delineation for a creek that transported naturally occurring asbestos and was subject to flooding/dredging events. Reviewed and critiqued USEPA sampling and risk evaluation. Key issues that affected interpretation of risk were related to the type of analysis performed in comparison with hose used to develop the standards.
- Risk Assessment of Arsenic in Gravel Products. Directed probabilistic risk assessment for potential exposures to arsenic in gravel used for road construction and landscaping.
- USDA Evaluation of Cadmium in Oysters, U.S. Pacific Coast (2008). Collaborated with a multidisciplinary team to evaluate potential risks associated with subsistence-level consumption of oysters collected in the Pacific Northwest. This project involved evaluation of federal and international tolerance limits for cadmium in oysters, shellfish ingestion rates for tribal and general populations, and bioavailability of cadmium in oysters.
- Evaluation of Dioxin Exposures from Dredged Material Disposal, Puget Sound, Washington. Provided comments for ports on an USEPA/US Army Corps of Engineers analysis of potential exposures to polychlorinated dibenzo-p-dioxins and dibenzofurans in seafood affected by dredged material disposal in Puget Sound.
- Risks of Residual Petroleum, Suquamish, Washington. Provided oversight for evaluation of potential exposures to residual petroleum at a tribal beach affected by a recent oil spill. Exposures due to consuming shellfish, harvesting aquatic vegetation, and performing other activities at the impacted beach were assessed. Shellfish bed reopening criteria were evaluated. A statistical comparison of oil-impacted and reference location shellfish tissue and sediment data was conducted.
- Evaluation of Arsenic in Fish. Performed a literature review and analysis of inorganic arsenic in seafood, with recommendations regarding the assumed fraction of total arsenic that is inorganic. Also supported an analysis of arsenic uptake in fish. Both studies were published.
- Dietary Intake of Arsenic. Directed the first U.S. market basket survey of inorganic arsenic in the diet. Findings were published.
- Risks of Herbicide Application to Lakes, Washington. For Washington State Department of Ecology, updated and revised a human health risk assessment for an EIS on the application of herbicides to Washington lakes.
- School Property Pesticide Evaluation, New York. For a New York State school district, evaluated potential exposures of students to pesticide residues in athletic field soils. Arsenic was of primary concern. Also evaluated potential association of lymphoma cases to pesticides detected in soil.
- School Property Evaluation, New York. On behalf of a school board in New York State, critically evaluated a state investigation of contamination at a high school built on a former industrial site. Presented findings at a public meeting of a citizen's advisory committee.
- Methylmercury Exposure Study, Nome, Alaska. For the Norton Sound Health Cooperative, participated in planning and design of a study of methylmercury concentrations in hair of native Alaskans subsisting on fish and sea mammals in Nome, Alaska.
- Risks of Road Fill, Alaska. Assisted the Alaska Department of Transportation as a subcontractor in a preliminary assessment of risks from metals and pesticides in fill material used during the construction of a road in Alaska.

Regulatory and Research Projects

- Microplastics guidance (2021-2022). Contributed to health risk assessment sections of ITRC (Interstate Technology & Regulatory Council (2023) guidance on microplastics. https://mp-1.itrcweb.org/
- Alabama Ambient Water Quality Criteria for Arsenic (2021). In support of the Alabama Mining Association's comments on proposed revisions to the Alabama Department of Environmental Management's arsenic water quality criteria for human health, provided comments on the assumed bioconcentration factor (BCF) used to predict fish tissue arsenic concentrations from water concentrations, a proposed adjustment to reflect the fraction of total arsenic in fish that is inorganic (i.e., the inorganic fraction), and updated information on arsenic toxicity.
- Regulation of Plastic Aquaculture Gear (2016-2017). Provided comments on a proposal by the City of Bainbridge, WA to ban plastic aquaculture gear and comments on the City submittal for a Shoreline Master Program Limited Amendment for aquaculture that proposes a ban on the use of non-biodegradable plastics in aquaculture operations. Comments addressed allegations of chemical and microplastic release from aquaculture gear.
- Soil PAH and PCB Relative Bioavailability (2016). Conducted a review of the relative bioavailability of PAHs and PCBs in soil for the Danish Environmental Protection Agency.
- Factors Affecting Exposure to Mercury in Fish (2016). Prepared review of factors affecting fish mercury exposure, including fish consumption, fraction of total mercury that is methylmercury, impacts of cooking on mercury concentration, and other factors. Confidential client.
- Fish Mercury Bioavailability (2015). Prepared review and analysis of factors affecting the bioaccessibility of mercury in fish, including variability by fish species, impacts of cooking, and of other foods. Confidential client.
- Health Canada Bioavailability Guidance (2010-2011 and 2014). Developed comprehensive "Guidance on Consideration of Oral Bioavailability of Chemicals in Soil for Use in Human Health Risk Assessment" and provided peer review for final streamlined guidance document. Focused on metals including arsenic, cadmium, chromium, lead, mercury, and nickel.
- Mercury Emissions (2011). Prepared comments on USEPA's December 2011 Technical Support Document: National Scale Mercury Risk Assessment Supporting the Appropriate and Necessary Finding for Coal- and Oil-Fired Electric Generating Units, focusing on assumptions about methylmercury uptake into fish and fish consumption rates, on behalf of Southern Company.
- Fish Consumption Rates (2011). Submitted comments on Washington Department of Ecology draft report titled Fish Consumption Rates Technical Support Document: A Review of Data and Information About Fish Consumption in Washington, dated September 2011, on behalf of the Pacific Coast Shellfish Growers Association.
- Arsenic Ambient Water Quality Criteria (2011-2012). Made presentations, gave testimony and submitted comments in support of increases in the Maine ambient water quality criteria (AWQC) for inorganic arsenic (health protection) to 1.2 µg/L for water and organisms and to 2.8 µg /L for organisms only, and to increase the arsenic screening standard for the agronomic utilization of sewage sludge (biosolids) from 10 mg/kg to 34 mg/kg, on behalf of the Arsenic Legislation Coalition.
- Dermal Absorption of Chemicals in Soil and Sediment (2011-2012). Directed preparation of a report to address uncertainties in dermal absorption of contaminants from soil and sediments. Health Canada may use the report in a protocol for development of sediment quality guidelines and in guidance being developed for evaluation of human health risks associated with exposure to contaminated sediments, on behalf of Health Canada.

- Provided peer review comments on the selection and derivation of bioaccessibility and bioavailability values for inorganic lead in the Draft Revised Health Canada Human Health Soil Quality Guidelines (2011), on behalf of Health Canada.
- SERDP Soil PAH Bioavailability Project. Participated in Department of Defense (DoD) Strategic Environmental Research and Development Program funded research project on PAH interactions with soil and effects on bioaccessibility and bioavailability to humans.
- SERDP Soil Metal Bioavailability Project. Participated in DoD Strategic Environmental Research and Development Program funded research project on soil metal bioaccessibility and bioavailability to humans.
- Bioavailability White Paper. Directed the development of a white paper for the Ontario Ministry of Environment on the use of oral bioavailability adjustments in a human health risk assessment. Peer-reviewed literature was critically reviewed and synthesized. The resulting white paper provides a background for use of bioavailability studies, site-specific and chemical-specific concerns, use of soil amendments to reduce bioavailability, methods for conducting in vitro and in vivo bioavailability studies, and other factors to consider when applying relative bioavailability adjustments in risk assessment.
- Arsenic Bioaccumulation and Speciation in Seafood. Conducted a literature review and prepared a document supporting USEPA re-evaluation of the ambient water quality criterion (AWQC) for human health effects of arsenic. Calculated AWQC using studies on bioconcentration factors and arsenic speciation acquired from the literature review. Directed the development of manuscripts evaluating bioaccumulation and speciation of arsenic in seafood.
- Regulatory Comment, California. Submitted comments on a California draft public health goal for arsenic in drinking water.
- New Facility Air Permit Support, Washington. On behalf of private developer, derived a risk-based acceptable new source impact level (ASIL) for use in evaluating predicted air releases of 1,3-butadiene from a proposed recreational facility in Washington.
- Department of Defense Bioavailability Field Guide. Updated a Department of the Navy Field Guide for use by Department of Defense project managers in evaluating the bioavailability of metals in soil to both ecological and human receptors at contaminated sites. Also contributed major sections of the original Field Guide.
- TPH Standards Guidance Review, West Virginia. Critically reviewed draft supplemental guidance on the development of total petroleum hydrocarbons risk-based standards for the West Virginia Department of Environmental Protection. Verified the applicability of analytical methods and TPH carbon range fractions proposed and evaluated appropriateness of toxicity factors developed in the guidance. Provided review comments in context of the TPH Criteria Working Group guidance documents and Massachusetts TPH risk policy.
- Metal Bioavailability Research Program. Managed a bioavailability research program of arsenic and lead in soils contaminated by mining and smelting wastes. Findings demonstrated the reduced absorption of these metals from soils. Mineralogic analyses and in vitro screening studies were used to help interpret the results of animal studies. Research results have been published in peer-reviewed journals and have been cited by USEPA in support of precedent-setting changes in risk assessment assumptions that resulted in much higher cleanup levels.
- Taiwanese Dietary Arsenic Research Project. Directed the investigation of inorganic arsenic intake in the diet of people living in areas of Taiwan that have elevated arsenic concentrations in artesian well water. Samples of rice and yams collected in Taiwan showed that arsenic intake from the Taiwanese diet was much higher than previously assumed, suggesting that USEPA's toxicity values might overestimate arsenic toxicity.

- US Dietary Arsenic Research Project. Directed an investigation of dietary arsenic intake in the US. A market basket survey of 40 commodities demonstrated the presence of inorganic arsenic as a normal occurrence in the American diet.
- Copper Smelter Risk Assessment Research Program, Montana. Provided strategic risk assessment support during an eight year period for the evaluation of four operable units at a former copper smelter site in Anaconda, Montana. A research program was developed to fully characterize potential risks associated with arsenic, cadmium, and lead in soil and waste materials from copper smelting operations. Participated in the review of work plans and data interpretation by a working group of client and USEPA staff and consultants. An epidemiology study demonstrated that current exposures were negligible, and studies of arsenic bioavailability and soil ingestion provided support for site-specific assumptions. Soil arsenic cleanup levels of 250, 500 and 1,000 mg/kg for residential, industrial and recreational areas, respectively, were adopted based on the application of these site-specific studies in the human health risk assessments for the site.
- Regulatory Comment. For an industry association, directed the preparation of comments on the Proposed Rule on the Bevill Exclusion for Mining Wastes. Critiqued USEPA's assessment of damages to human health and the environment caused by land-based units, and concluded that most of the damages cited by USEPA are only of historical relevance and do not reflect current mining practices. Also critiqued the use of the toxicity characteristic leaching procedure as a means of measuring the toxicity of mineral processing wastes, and concluded that it is an overly aggressive and unrealistic test for evaluating these materials.
- Sub-title D Municipal Landfill Permitting, New Mexico. For client attempting to site a subtitle D municipal landfill, prepared an affidavit rebutting assertions regarding potential adverse health effects of such landfills. Affidavit was submitted to the docket for the permit hearing and resulted in withdrawal of the allegations.
- Evaluation of Ocean Disposal of Dioxin-Containing Sediments, Grays Harbor, Washington. For the US Army Corps of Engineers, performed a risk assessment for ocean disposal of dioxin-contaminated sediments from Grays Harbor, Washington. Evaluated potential exposures to dioxins in Dungeness crabs that might contact contaminated sediments in an ocean disposal site for dredged materials. Evaluation included derivation of site-specific crab consumption values and crab life cycle evaluation.
- PCB Risk Assessment Sensitivity Analysis. Directed a PCB risk assessment sensitivity analysis project. Identifying those components of risk assessment methodology that have the greatest influence on PCB cleanup levels.
- EIS Health Impact Analysis, Washington. For the Washington State Department of Ecology, evaluated the potential human health impacts of cleanup alternatives for an EIS for State Model Toxics Control Act. Participated in developing the risk-based alternative.

Litigation Projects

- Geoduck Farm Hearings (2024). Provided testimony related to the use of plastic aquaculture gear at the Burley Lagoon FEIS Appeal hearing and the related permit hearing. Appeal of Coalition to Protect Puget Sound Habitat, Friends of Burley Lagoon, Tahoma Audubon Society, and Friends of Pierce County of the January 6, 2023 Burley Lagoon Geoduck Farm Final Environmental Impact Statement issued by Pierce County. Appeal No. 1004944, SD/CP 15-14.
- Uranium Processing Facility Litigation (2024). Provided an expert report and deposition testimony on behalf of Honeywell International Inc. in the following matters related to Honeywell's Metropolis Works Uranium Conversion Facility (MTW) in Metropolis, IL: Dassing v. Honeywell (Consolidated Case No. 3:21-cv-00485), Steward v. Honeywell (Case No. 3:18-cv-01124), Metropolis and Massac County v. Honeywell (Case No. 3:21-cv000860).

- International Arbitration (2024). Provided expert testimony under the rules of the United Nations Commission on International Trade Law related to exposure to metals released by a smelter in South America in an arbitration. The Renco Group, Inc., and Doe Run Resources Corp. v. The Republic of Peru and Activos Mineros S.A.C. ("PCA Case No. 2019-47").
- PFAS Multidistrict Litigation (confidential, protective order) (2022). Provided expert reports and deposition testimony in Re: Aqueous Film Forming Foams Products Liability Litigation, multidistrict litigation No. 2:18-MN-2873-RMG, U.S. District Court for the District of South Carolina, Charleston Division.
- Coal Combustion Residuals Insurance Litigation (confidential, protective order) (2022). Provided expert reports and deposition testimony in AEP Generation Resources Inc., et al. v. AG Insurance SA/NV et al., Case No. 18CV004317, in the Court of Common Pleas, General Division, Franklin County, Ohio.
- Lead Personal Injury Litigation (2022). Provided expert reports and trial testimony in the matter of Sanders vs. Mount Isa Mines Ltd before the Supreme Court of Queensland, Australia (Registry: Brisbane, Proceeding No.: 7608/11).
- Municipal Solid Waste Litigation (confidential) (2021). Provided opinions related to potential exposures to metals in ash produced by solid waste incineration.
- Lead Paint Personal Injury Litigation (confidential) (2021). Provided expert opinions for a case alleging harm to a child from lead paint in a rental property. Case settled.
- International Arbitration (confidential) (2014 and 2020). Provided expert opinions related to exposure to metals released by a smelter in South America.
- PCB Tort Litigation (2018-2021). Providing expert opinions regarding PCB exposures in a school. Bard et al. v. Monsanto et al. Case No. 18-2-00001-7. Superior Court of Washington for King County.
- Coal Ash Basin Litigation (2020). Provided an expert rebuttal report and deposition testimony regarding historical knowledge of toxicity and health risks associated with coal ash basins at 14 coal-fired electricity generating plants in the matter of 17-CVS-5594, Duke Energy Carolinas, LLC and Duke Energy Progress, LLC v. AG Insurance SA/NV et al., in the Superior Court Division of the North Carolina General Court of Justice. Case settled.
- Coal Ash Basin Litigation (2019). Began developing expert opinions regarding possible health risks for various scenarios for managing coal ash basins. Duke Energy Carolinas LLC v. State of North Carolina ex rel. North Carolina Department of Environmental Quality, et al. Case Nos. 19 EHR 2398, 19 EHR 2399, 19 EHR 2401, 19 EHR 2403, 19 EHR 2404, 19 EHR 2406, North Carolina Office of Administrative Hearings. Case settled.
- Cleaning Fluid Exposure Litigation (2018). Provided expert report regarding exposures to chemical mixtures in cleaning fluid added to a water bottle. Teepe and Teepe v. State of Washington Department of Corrections, Anthony Anfinson. Case No. 17-2-00470-8. Superior Court of the State of Washington in and for Walla Walla.
- Concrete Sealant Fume Litigation (2018). Provided expert opinions regarding exposure and toxicity of odorant chemicals released from driveway sealant product. Kaiser Foundation Health Plan v. Quality Design/Build Inc. Case No. 16-1-0987-05 JHC and Kobayashi v. Quality Design/Build Inc. Case No. 16-1-0985-05 KKS in the Circuit Court of the First Circuit, State of Hawaii. Cases settled.
- Lead Exposure Litigation (2016-2020). Provided expert opinions regarding lead exposures in Flint to Michigan Attorney General's Office. Multiple cases.

- Lead Paint Complaint (2017). For the defense, provided an expert report and testimony at an evidentiary hearing about peeling lead paint on an elevated subway line in Queens, NY. Dudley Stewart, et al. vs. the Metropolitan Transit Authority, et al. Case No. 17-CV-03060 (RJD). U.S. District Court Eastern District of New York. December 2017. Case settled.
- Coal Ash Basin Litigation (2017-2018). Provided an expert report and deposition testimony regarding any health risks that might be associated with the coal ash basin at the Mayo Steam Station coal-fired electricity generating plant in Person County, NC. Roanoke River Basin Association vs. Duke Energy Progress LLC. Case No. 1:16-cv-607. U.S. District Court for the Middle District of North Carolina.
- Coal Ash Basin Litigation (2017-2018). Provided an expert report and deposition testimony regarding any health risks that might be associated with the coal ash basin at the Roxboro Steam Station coal-fired electricity generating plant in Person County, NC. Roanoke River Basin Association vs. Duke Energy Progress LLC. Case No. 1:17-cv-452. U.S. District Court for the Middle District of North Carolina.
- Mine Operations Litigation (confidential) (2017). In connection with an investigation of a spill of leach solution from a high altitude gold mine in South America, prepared an assessment of potential downstream health risks.
- Shoreline Development Permit State Appeal, Washington (2016). Provided testimony before the Thurston County Hearing Examiner at a hearing appealing the determination of mitigated non-significance in the request of Chang Mook Sohn for substantial shoreline development permit for an intertidal geoduck aquaculture operation. Testimony addressed claims asserted by Appellants' experts regarding potential for adverse impacts of plastic gear used in geoduck aquaculture, including potential release of microplastics. Project No. 2014108800. October 2016.
- Appeal of Permit for Control of *Zosteria Japonica* on Commercial Clam Beds, Washington (2015). Provided testimony at a hearing appealing the issuance of a NPDES and state waste discharge general permit (October 2015). Testimony addressed claims asserted by Appellants' experts regarding potential for adverse toxicological and environmental impacts of spraying the herbicide imazamox on Japanese eelgrass beds. Pollution Control Hearings Board, Case reference PCHB No. 14-047. "Coalition to Protect Puget Sound, Ross P. Barkhurst, Robert Kavanaugh v. Ecology and Willapa-Grays Harbor Oyster Growers Association."
- Shoreline Development Permit State Appeal, Washington (2015). Provided testimony at a hearing appealing the issuance of the Shoreline Development Permit (SD5-13) (March 2015). Testimony addressed claims asserted by Appellants' experts regarding potential for adverse impacts of plastic gear used in geoduck aquaculture, including potential release of microplastics. Shorelines Hearings Board, State of Washington, Case reference SHB No. 14-024. "Coalition to Protect Puget Sound Habitat vs. Pierce County, Taylor Shellfish and Seattle Shellfish."
- Former Shooting Range Litigation, California (2013). For the defense, provided expert opinions at deposition related to potential lead exposures at a former shooting range. Otay Land Company vs. U.E. Limited, L.P. Superior Court of the State of California, Case No.: GIC869480 and Case No.: 37 -2009 -0010197 6-CU-OR-CTL [Consolidated for all purposes] in and for the County of San Diego. March 2013.
- Provided consultant and expert witness services related to alleged coal tar sealant exposure for the matter of Joseph L. Hatchell vs. SealMaster, Inc., Tony Shelley and SealMaxx, Inc. State of South Carolina, County of Darlington Common Pleas Court. 2013-2014.
- Clean Water Act Violations. Provided expert opinions and trial testimony related to potential chlorine gas exposures for the defense (Federal Public Defender) in US v. Patrick Dooley, Case No. CR11-252MJP, US District Court, Western District of Washington at Seattle. January 2012.

- Shoreline Development Permit State Appeal, Washington. Provided testimony at a hearing appealing county determination of nonsignificance for a shoreline substantial development application for a shellfish-geoduck farm (March 1, 2012). Testimony addressed claims asserted by Appellants' experts regarding the toxicity of metals in polyvinyl chloride (PVC) tubing used in geoduck aquaculture. Shorelines Hearings Board, State of Washington, Case reference SHB No. 11-019. "Coalition to Protect Puget Sound Habitat and Case Inlet Association vs. Pierce County and Longbranch Shellfish LLC." 2012.
- Shoreline Development Permit County Appeal, Washington. Provided expert report and testimony at a hearing appealing county determination of nonsignificance for a shoreline substantial development application for a shellfish-geoduck farm (March 2011). Report and testimony addressed claims asserted by Appellants' experts regarding the toxicity of metals in polyvinyl chloride (PVC) tubing used in geoduck aquaculture. Pierce County Hearing Examiner case reference SD_22-06\688646, "Coalition to Protect Puget Sound Habitat vs. Longbranch Shellfish LLC." 2011.
- Evaluation of Coke Plant Emissions. Provided expert report regarding the potential for increased health risks due to SO_2 and $PM_{2.5}$ among residents and employees of the city of Monroe due to air emissions from the Middletown Coke Company Plant. Ohio Environmental Review and Appeals Commission. Robert D. Snook Natural Resources Defense Council et al., City of Monroe, Ohio v. Chris Korleski, Middletown Coke Company, Inc., SunCoke Energy, Inc. Case Nos. ERAC 096432-8. 2011.
- Indoor Air Quality. Provided a declaration in support of motion to strike opposing expert's declaration related to a residential indoor air quality evaluation in April Norton Rauch v. Ballard Leary Phase II, LP, BRCP/CPI Leary LLC, YOTM LLC, No. 09-2-21126-4 SEA in the Superior Court of the State of Washington, King County. 2011.
- Worker Compensation Case, Washington. Provided expert opinions and deposition for the defense for an appeal to the Board of Industrial Insurance Appeals regarding alleged solvent exposures and toxic solvent encephalopathy and Alzheimer's disease. Claim No. SB-29840. 2010.
- Insurance Cost Recovery Litigation, Multiple Sites. Provided expert opinions and deposition regarding historical knowledge of arsenic toxicity in a case related to remediation of arsenical pesticide production/formulation facilities. U.S. Borax Inc. v. Royal Indemnity Company, et al. Case settled. 2009.
- Provided expert opinion (report and declaration) regarding plaintiff's allegations of adverse health effects resulting from releases of arsenic and other chemicals from the FMC Corporation Plant in Middleport, New York. Lewis, et al. v. FMC Corporation. Civil Docket No.: 1:04-CV-00331-WMS. United States District Court Western District of New York. 2007.
- Mining and Smelting Site Litigation, Idaho. Provided expert report for the defense regarding class action allegations of proposed medical-monitoring classes related to lead exposures identified in the Second Amended Class Action Complaint ("Complaint") for Baugh v. Asarco. Case No. CV 02-00131. Certification denied. 2004.
- Mining Site Litigation, Oklahoma. Provided expert opinions and deposition for the defense regarding lead exposures in an historic zinc mining district. Betty Jean Cole, et al. Plaintiffs, vs. ASARCO Incorporated, et al., Defendants; The Doe Run Resources Corporation, Third Party Plaintiff, vs. United Sates of America, Third Party Defendant. United States District Court for the Northern District of Oklahoma. Case No. 03-CV-327-H(C). Class certification ruling resolution unknown. 2004.
- Copper Chromated Arsenic Litigation, Indiana. Provided expert opinions for the defense regarding copper chromated arsenic (CCA) exposures from a residential deck. James R. Hayden and Jamie F. Hayden, Husband and Wife, v. Menard, Inc. et al. Case No. 1:03-cv-1400-DFH-TAB. Case settled. 2005.

- Roxarsone Litigation, Arkansas. Provided expert opinions and deposition for the defense regarding exposures to roxarsone from poultry operations. Court sustained defendants' motion for summary judgment. Mary E. Green, et al. v. Alpharma Inc., et al. Case No. CIV-03-2150-2. Circuit Court of Washington County, Arkansas. 2005-2006.
- Asbestos Litigation, Washington. Provided expert opinions and deposition for the plaintiffs regarding exposures to asbestos-containing dusts by apartment tenants occupying a multiunit residential building during a large-scale plumbing renovation that occurred in the absence of asbestos control measures. Franklin Albano; et al. v. Scott Real Estate Investments, Inc., et al. Superior Court for the State of Washington in and for King County. No. 04-2-12469-7 SEA. Case settled. 2006.
- Residential Mold Litigation, Washington. Provided expert opinions for the defense regarding mold exposures in a tenant/landlord dispute. Helms v. Sharma. Snohomish County Superior Court. Case No. 03-2-06536-4. Case settled. 2005.
- Copper Mining District Releases, Utah USA. Prepared deposition describing limitations of potential adverse effects associated with lead and arsenic in a riverbed near a residential area downstream of a mining site in Utah. Deposed by US Department of Justice and USEPA attorneys, and testified for an alternative dispute resolution. 1990s.
- Vapor Intrusion Litigation, Seattle, Washington. Named as testifying expert and prepared for deposition in litigation related to potential impacts of infiltration of vapors from volatile chemicals in groundwater into a business adjacent to a waste chemical processing facility. Conducted an in-depth review of baseline risk assessment, inhalation pathway assessment, and state health consultation prepared for site; evaluating building vapor intrusion modeling; critically assessing USEPA's Johnson and Ettinger model; assessing potential employee exposures; and researching toxicity assessments of chlorinated volatile organic chemicals and benzene. Compiled list of technical questions to be asked of opposing expert. 1990s.
- Pesticide Manufacturing Facility Releases, Texas. Made a presentation to a district attorney and investigators conducting a manslaughter investigation related to chemical releases from a pesticide manufacturing facility in Texas. 1990s.

CAREER

2010-Present
Ramboll US Consulting, Inc./ENVIRON US Corp., Seattle, Washington
Principal

2002-2010
Integral Consulting, Inc., Mercer Island and Seattle, Washington
Principal

2000-2002
Gradient Corporation, Mercer Island, Washington
Principal

1988-2000
PTI/Exponent, Bellevue, Washington
Principal and Senior Toxicologist

1987-1988
Environmental Toxicology International, Seattle, Washington
Senior Toxicologist

1982-1987

Ortho Pharmaceutical Corporation (Drug Safety Evaluation), Raritan, New Jersey
Toxicologist

1975-1977

US Environmental Protection Agency (Office of Toxic Substances), Washington, DC
Chemist

PEER-REVIEWED PUBLICATIONS AND BOOK CHAPTERS

Schoof RA, Van Landingham C, Bailey A. 2022. Assessing the Impact of a Residential Metals Abatement Program on Child Blood Lead levels in Butte, MT. Society for Mining, Metallurgy and Exploration (SME) journal.

Van Landingham, C., Fuller, W.G., Schoof, R.A. 2020. The effect of confounding variables in studies of lead exposure and IQ. Crit Rev Tox. DOI: 10.1080/10408444.2020.1842851.

Tu, J.W., Fuller, W., Feldpausch, A.M., Van Landingham, C., Schoof, R.A. 2020. Objective ranges of soil-to-dust transfer coefficients for lead-impacted sites. Environ Res 184: 109349. PMID: 32199320 DOI: 10.1016/j.envres.2020.109349.

Feldpausch AM, Rodricks JV, Schoof RA, Weldon BA. 2018. Gastrointestinal tract development and its importance in toxicology. Toxicology of the Gastrointestinal Tract, Second Edition, Ed. Shayne Cox Gad. CRC Press. ISBN 9781138360167.

Schoof RA & DeNike J. 2017. Microplastics in the context of regulation of commercial shellfish aquaculture operations. Integrat Environ Assess Manage 13:522–527.

Schoof RA & Handziuk E. 2016. Arsenic speciation and bioavailability in vegetables. In Arsenic Research and Global Sustainability. Battacharya P, Vahter M, Jarsjö J, Kumpiene J, Ahmad A, Sparrenbom C, Jacks G, Donselaar E, Bundschuh J, and Naidu R. (eds). pp313-315. ISBN 978-1-138-02941-5.

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Steele MJ, Whittaker SG and Schoof RA. 1992. The impact of assumptions regarding metal concentrations in soil and dust on setting remedial objectives. In: Risk assessment/management issues in the environmental planning of mines. Van Zyl D, Koval M and Li TM (eds.) Society for Mining, Metallurgy and Exploration, Littleton, CO.

Davis A, Ruby MV and Schoof R. 1992. Comments on "Lead, cadmium, and zinc contamination of Aspen garden soils and vegetation," by Boon DY and Saltanpow PN. J Environ Qual 21:82–86, 509–510.

Katz LB, Schoof RA and Shriver DA. 1987. Use of a five-day test to predict the long-term effects of gastric antisecretory agents on serum gastrin in rats. J Pharmacological Methods 18(4):275–282.

Schoof RA and Baxter CS. 1986. Topical application of a tumor promoter induces proliferation of an adherent cell population in murine spleen. Int J Immunopharm 8:455–462.

Hahn DW, Hetyei N, Beck L, McGuire JL and Schoof RA. 1985. Pharmacology and toxicology studies with microencapsulated norgestimate as a long acting injectable contraceptive. Adv Contraception 2:235–236.

Baxter CS, Schoof RA and Lawrence AT. 1984. Interaction of tumor promoting agents with immunofunctional cells *in vitro* and *in vivo*. International Agency for Research on Cancer Scientific Publications, No. 56.

INVITED PRESENTATIONS/ PANELS/ PEER-REVIEWS/ AWARDS

2021 to 2022—Member of Interstate Technology & Regulatory Council (ITRC) team writing guidance on assessing environmental contamination by microplastics.

9/16 to 2020—Member of Strategic Environmental Research and Development Program (SERDP) Science Advisory Board. Appointment effective June 2017-June 2020. SERDP is one of two U.S. Department of Defense environmental research programs, harnessing the latest science and technology to improve DoD's environmental performance, reduce costs, and enhance and sustain mission capabilities. Activities included review of projects assessing polyflourinated alkyl substance (PFAS) contamination and replacement firefighting foams, impacts of climate change on threatened and endangered species, unexploded ordinance, bilge water, waste management at field bases and other technology issues.

4/20—External peer review of a report summarizing an evaluation of the Integrated Exposure Uptake Biokinetic (IEUBK) Model (version 2.0) for U.S. Environmental Protection Agency (EPA), "Advancing Pb Exposure and Biokinetic Modeling for USEPA Regulatory Decisions and Site Assessments using Bunker Hill Mining and Metallurgical Complex Superfund Site Data".

12/06 to present—Human and Ecological Risk Assessment: Member editorial review board.

1/04 to present—Science Advisory Board for Contaminated Sites in British Columbia: Appointed member, Vancouver, BC.

07/19—Gave talk titled "Implications of the Threshold Approach for Food Safety Evaluation" in a workshop titled "Threshold-based Cancer Risk Assessment for Non-genotoxic Carcinogens: Inorganic Arsenic as a Case Study" at the Toxicology Forum conference in Alexandria, VA.

05/19—Gave keynote talk titled "Developing Oral Bioavailability Data for Health Risk Assessments" at the 15th International Conference on the Biogeochemistry of Trace Elements in Nanjing, China.

11/18—Gave talk titled "Assessing Soil Lead Exposures and Effectiveness of Remediation" at the BP 2018 Remediation Engineering & Technology Summit. Denver, CO.

10/18—Gave talk titled "Factors Affecting Mercury Exposure from Fish: Focus on Selenium/Mercury and Methylmercury/Total Mercury Ratios" at the Electric Power Research Institute Energy and Environment Program Fall Advisory Meeting. Denver, CO.

3/18—Gave talk titled "Factors Affecting Mercury Exposure from Fish" at the Electric Power Research Institute Energy and Environment Program Winter Advisory Meeting. Tampa, FL.

8/17—Gave talk titled "Factors Affecting Mercury Exposure from Fish" at the 13th International conference on Mercury as a Global Pollutant. Providence, RI.

9/17—Gave talk titled "Does plastic shellfish gear increase microplastic and chemical exposures?" at the annual meeting of the Pacific Coast Shellfish Growers' Association. Welches, OR.

1/17—Gave talk titled "Human health risk assessment for mercury at sediment sites" at a workshop of the Ninth International Conference on Remediation and Management of Contaminated Sediments, New Orleans.

2/16—Gave talk titled "Factors Affecting Fish Mercury Bioavailability" at the Electric Power Research Institute Winter Environment Program Advisory and Sector Council Meetings. Phoenix, AZ.

2016—Served as thesis examiner for R Lewatu Taga. Revised thesis titled "Development of in-vitro methods to predict bioavailability of arsenic, cadmium, copper, lead and zinc in mine wastes for human health risk assessment", submitted for a degree of Doctor of Philosophy at the University of Queensland, Sustainable Minerals Institute, 2016.

11/14 to 2/15— External Peer Review of EPA's Approach for Estimating Exposures and Incremental Health Effects due to Lead from Renovation, Repair, and Painting Activities in Public and Commercial Buildings. Served as one of 12 appointed peer reviewers.

11/13 to 7/14—Washington Department of Ecology PCB Chemical Action Plan Advisory Committee. Served on committee reviewing state-wide chemical action plan.

2014—Served as thesis examiner for R Lewatu Taga. Thesis titled "Development of in-vitro methods to predict bioavailability of arsenic, cadmium, copper, lead and zinc in mine wastes for human health risk assessment", submitted for a degree of Doctor of Philosophy at the University of Queensland, Sustainable Minerals Institute, 2014.

2009 to 2013—Washington Department of Ecology MTCA Science Panel. Appointed member of Model Toxics Control Act Science Panel.

2013—International Society for Exposure Science meeting in Seattle, WA. Served on the organizing committee, including conducting reviews of session proposals and abstracts.

2013—Served as thesis examiner for J Zheng. Thesis titled "Lead from Mining and Mineral Processing Activities to the Community via the Air-dust Pathway: An Example from Mount Isa City Using Human Health Risk Assessment Approach", submitted for a degree of Doctor of Philosophy at the University of Queensland, Sustainable Minerals Institute, December 2012.

10/12 – Gave an invited talk titled "Low Lead Level Exposures Today" at HB Lead Litigation Conference, New Orleans, October 2012.

9/12—Fish Mercury Bioavailability, (with a short arsenic detour). Electric Power Research Institute Fall Environment Program Advisory Meetings. P42 – Air Toxics Council Meeting. Milwaukee, WI.

7/12—Synergies of HIA and Ecosystem Services in International Development Projects. Co-organizer of session. Talk presented titled "Health Impact Assessment – A Key to Sustainable Development" at the World Congress on Risk in Sydney, Australia. Session co-chair.

3/12—Beyond Lead and Arsenic: How are other metals being addressed? Invited talk presented at workshop session titled "Assessing the Bioavailability and Risk from Metal-contaminated Soils and Dusts" at the 51st Meeting of the Society of Toxicology in San Francisco, CA.

2011—Served on thesis examination committee for W. Cutler. Thesis titled "Bioaccessible arsenic in soils of the Island of Hawaii. A dissertation submitted to the graduate division of the University of Hawai'i at Mānoa in partial fulfillment of the requirements for the degree of doctor of philosophy in geology and geophysics.

9/09—5th International Workshop on Chemical Bioavailability, Adelaide, Australia. Invited plenary lecture titled "Developing Oral Bioavailability Data for Risk Assessments: Method Development and Burden of Proof".

1/06 to 1/09—Metals in the Human Environment Research Network (a Canadian university research consortium funded by the Canadian National Science and Engineering Research Council): Expert advisory panel member, Gatineau, Quebec.

1/09—National Research Council: Peer review of "Contaminated Water Supplies at Camp Lejeune—Assessing Potential Health Risks," November 2008 Peer Review Copy.

10/08 to 6/09—DuPont-USEPA PFOA Peer Consultation Panel. Peer consultation on exposure assessment for perfluorooctanoic acid released from DuPont Washington Works facility.

1/07 to 07/08—National Research Council: Committee on Beryllium Alloy Exposures, Washington, DC.

10/07—Lifetime Achievement Award given by the Annual International Conference on Sediments Soils and Water under the auspices of the University of Massachusetts for significant contribution to a field of science or engineering, as assessed by the level and longevity of contributions, assumption of responsibilities, and volunteerism for charitable organizations and not-for-profit groups.

10/07—International Society for Exposure Analysis annual meeting: Co-organizer of symposium titled "Use of In Vitro Bioaccessibility/Relative Bioavailability Estimates in Regulatory Settings: What Is Needed?" and talk titled "Method development and the application of oral bioavailability data in US risk assessments," Durham, NC.

8/06 to 12/06—Toxicology for Excellence in Risk Assessment: Peer review panel member for review of the Sudbury Soils Study Human Health Risk Assessment, Sudbury, Ontario.

9/06—International Society for Exposure Analysis annual meeting: Invited speaker at symposium titled "Childhood exposures to bioavailable metals in soil and household dust in residential environments," talk titled "Method development and the application of oral bioavailability data in US risk assessments," Paris, France.

9/06—American Chemical Society annual meeting: Invited speaker at Agricultural & Food Chemistry Division symposium on Heavy Metals in Food, talk titled "Dietary intake of toxic forms of arsenic," San Francisco, CA.

9/06—Electric Power Research Institute: Invited speaker at Environment Sector meeting, talk titled "Critical evaluation of ambient water quality criterion for arsenic: bioaccumulation and speciation issues," Atlanta, GA.

11/05—University of Washington Department of Environmental Health seminar series: Invited lecture titled "Probabilistic lead model for an operating smelter in South America," Seattle, WA.

6/05—XIII International Conference on Heavy Metals in the Environment: Invited member of Arsenic Environmental Health Research Panel, talk titled "Arsenic speciation in commonly consumed organisms," Rio de Janeiro, Brazil.

6/04—Canadian National Science and Engineering Research Council: Research grant application site visit panel member, Guelph, Ontario.

3/04—Society of Toxicology annual meeting: Co-chair of risk assessment poster session, Baltimore, MD.

2/04—US Environmental Protection Agency: Peer review of Lead Bioavailability Technical Support Document.

1/04—Sustainable Land Application Conference: Invited lecture titled "The evolving science of chemical risk assessment as applied to land application of biosolids effluents and manures," Lake Buena Vista, FL.

8/02 to 3/04—National Research Council: Member of Subcommittee on Toxicological Risks to Deployed Military Personnel.

11/03—People to People Ambassador Program: Toxicology Delegation to China: Beijing, Guilin, and Shanghai.

4/03—US Environmental Protection Agency: Invited Speaker at workshop on bioavailability of metals in Tampa, FL. Gave talk on the role of bioavailability model validation in site-specific decision-making.

7/02—National Research Council: Peer review of "Bioavailability of contaminants in soils and sediments: Processes, tools and applications," Peer Review Copy.

8/02—US Environmental Protection Agency: Peer review of draft "Estimates of soil ingestion in children," by Cain et al.

6/02—Mealey's Emerging Toxic Tort Conference: Lecture titled "Up to date analysis of water contamination cases: The science," Pasadena, CA.

3/01 to 6/02—National Research Council: Appointment to Committee on Toxicants and Pathogens in Biosolids. Book issued titled Biosolids applied to land: advancing standards and practices. Participated in Congressional briefing of committee findings.

4/02—Center for Environmental & Occupational Risk Analysis and Management, College of Public Health, University of South Florida, Tampa: Lecture titled "Consideration of background exposures and bioavailability in designing arsenic biomonitoring studies."

3/02—Electric Power Research Institute Advisory committee meeting. Lecture titled "Arsenic exposure and risk: Public perception vs. likely exposure pathways."

10/01—Ontario Ministry of the Environment. Member of international peer review panel evaluating draft risk assessment for the Rodney Street community in Port Colborne, FL. Participated in media briefing and community open houses to explain role of peer review panel. Named by the Ontario Ministry of the Environment as a testifying expert for the executive review tribunal.

10/01—Contaminated Soils, Sediments and Water annual conference: Lecture titled "Methodological issues in assessing dermal absorption of chemicals" in Dermal Bioavailability session.

9/01—Northwest Biosolids Management Association annual conference: Keynote speech describing the National Research Council biosolids committee membership and charge.

4/01—US Environmental Protection Agency: Peer review of draft supplemental guidance for developing soil screening levels for Superfund sites.

3/01—Society of Toxicology annual meeting: Lecture on metal bioavailability in continuing education course on risk assessment of metals.

3/01—Society of Toxicology annual meeting: Co-chair of workshop on consideration of bioavailability in risk assessment.

2/01—Secretary of the Navy Environmental Awards FY 2000: Judge.

9/00—Agency for Toxic Substances and Disease Registry: Technical review of mercury releases from lithium enrichment at the Oak Ridge Y12 plant, July 1999.

9/00—US Environmental Protection Agency: Peer review of draft documentation for short-term arsenic toxicity value.

5/00—Agency for Toxic Substances and Disease Registry: Peer review of draft toxicity profile for creosote.

10/99—15th Annual International Conference on Contaminated Soils and Water (AEHS): Organized 3-hour workshop (taught with two colleagues) titled "Development of site-specific bioaccessibility and bioavailability data and their application to human health risk assessment." Co-organized and co-chaired technical session titled "Bioavailability of contaminants in soil," Amherst, Massachusetts, October 1999.

10/99—National Institute of Environmental Health Sciences Superfund Basic Research Program grant application review, special emphasis panel member, Research Triangle Park, North Carolina, October 1999.

7/99—ASCE-CSCE Environmental Engineering Conference: "Application of bioavailability to environmental cleanup settings: case studies," Norfolk, Virginia, July 1999.

5/99—Chemical Manufacturer's Association Exposure Assessment Workshop: Member of panel making recommendations regarding research projects CMA should fund in the area of dermal exposure assessment, Research Triangle Park, North Carolina, May 1999.

5/99—US Dept. of the Navy Remediation Innovation Technology Seminar series: One of four primary speakers for day long course. Topic was "The role of bioavailability in risk assessment," San Diego and San Francisco, California, Silverdale, Washington, Philadelphia, Pennsylvania, Charleston, South Carolina, and Honolulu, Hawaii, May 1999.

12/98—US Environmental Protection Agency workshop on issues associated with dermal exposure and uptake: Peer consultant reviewing draft risk assessment guidance, Bethesda, Maryland, December 1998.

12/98—National Environmental Policy Institute Conference-Bioavailability: Using what we know, learning what we need: "Why consider bioavailability in risk assessment?" Washington, D.C., December 1998.

8/98—US Environmental Protection Agency Modeling Lead Exposure and Bioavailability Workshop: "Interpreting *in vitro* bioavailability studies," Durham, North Carolina, August 1998.

7/98—Third International Conference on Arsenic Exposure and Health Effects: "A market basket survey of inorganic arsenic in food," San Diego, California, July 1998.

12/97—IBC's International Congress on Human Health Bioavailability: "Practical experience in developing/negotiating the use of bioavailability adjustments," Scottsdale, Arizona, December 1997.

9/96—US Environmental Protection Agency and US Department of Energy Mercury Speciation Workshop: "Biological models to predict soil mercury bioavailability to humans," Denver, Colorado, September 1996.

12/96—US Geological Survey Arsenic Workshop: "The role of bioavailability studies in deriving risk-based cleanup levels for arsenic in soil," Sutter Creek, California, December 1996.

3/96—NJDEP Interagency Risk Assessment Committee: "Assessing the oral bioavailability of metals in soil," Trenton, New Jersey, March 1996.

8/95—ATSDR Science Panel on the Bioavailability of Inorganic Mercury: Served as a member of an Agency for Toxic Substances and Disease Registry (ATSDR) expert science panel on the bioavailability of mercury in soil. Served as lead author on a manuscript reviewing methods and available data for assessing the oral absorption of various forms of inorganic mercury, Atlanta, Georgia, August 1995.

12/95—TNRCC Arsenic Symposium: Served as one of four invited experts at a 1-day symposium to brief toxicologists and project managers from the Texas Natural Resource Conservation Commission on the latest developments in assessing risks associated with arsenic in soil, Austin, Texas, December 1995.

1992—Oregon DEQ Cross Media Advisory Committee: Appointed by the Director of the Oregon Department of Environmental Quality (DEQ) to serve on an advisory committee that reviewed and commented on the methodology developed by DEQ to evaluate cross media regulatory impacts and develop a more integrated approach to the permit process. Also participated in technical subcommittee of toxicologists that provided detailed technical review of a comparative risk assessment model developed to rank chemical exposure and hazard to human and ecological receptors, Portland, Oregon, 1992–1993.

PRESENTATIONS & POSTERS

Schoof R, Schafer B, Linton K, Dally K. 2023. Relative Bioavailability Adjustment of Soil Arsenic Protective Concentration Level (PCL) at a Bryan, TX Site. AEHS East 2023 conference. Amherst, MA. (presented by K Dally).

Bailey A, Schoof R, Tu J. 2023. Relevance of Lead Enrichment in Soil Particle Size Fractions for Assessing Exposure. Toxicologist, Suppl Toxicol Sci 192 (1), abstract #3833.

Schoof RA, Van Landingham C, Bailey A. 2022. Assessing the Impact of a Residential Metals Abatement Program on Child Blood Lead levels in Butte, MT. Society for Mining, Metallurgy and Exploration (SME). Salt Lake City, UT. (presentation by A Bailey).

Schoof RA. 2020. Dietary arsenic risk assessment and risk management. Toxicologist, Suppl Toxicol Sci 174 (1), abstract #2218.

Van Landingham C, Schoof RA. 2020. Assessing the effectiveness of a residential metals abatement program (RMAP) using blood lead levels in children. Toxicologist, Suppl Toxicol Sci 174 (1), abstract #2847.

Feldpausch A, Schoof RA. 2020. Proposal for use of in vitro bioaccessibility data when methods validated using animal models are unavailable. Toxicologist, Suppl Toxicol Sci 174 (1), abstract #1524.

Schoof RA and Goodrich-Mahoney J. 2017. Factors affecting mercury exposure from fish. 13th International Conference on Mercury as a Global Pollutant. Providence, RI.

Schoof RA and Handziuk E. 2016. Arsenic speciation and bioavailability in vegetables. 6th International Congress on Arsenic in the Environment (As2016): Arsenic Research and Global Sustainability, Stockholm, Sweden. June.

Schoof RA. 2015. Children's blood lead levels in a mining community after 20 years of remediation activities. Society for Environmental Geochemistry and Health annual meeting, Arlington, TX. March.

Yager JW, Greene T, Schoof R. 2015. Arsenic relative bioavailability from diet and airborne exposures: Implications for risk assessment. Society of Toxicology annual meeting, San Diego, CA.

Schoof RA, Johnson DL, McConnell ER, Gallagher A, Van Landingham C, Feldpausch A and Dell L. 2014. Development of a Reference Dataset for Evaluation of Blood Lead Levels from Children in a Mining Community. International Society of Environmental Epidemiology annual meeting, Seattle, WA

Schoof RA, Johnson DL, Feldpausch A, McConnell ER, Van Landingham C, Dell L, Das M and Gallagher A. 2014. Characterization of blood lead levels for children in a community affected by historical mining activities. Society of Toxicology annual meeting, Phoenix, Arizona.

Goldsworthy B, Schoof R, Kaden D and Harris A. 2014. Hydraulic fracturing and human health concerns: Lessons from the US and Canada. Ecoforum Conference & Exhibition, Gold Coast, Australia.

Goldsworthy B and Schoof R. 2013. Assessment of mutagenic carcinogens in Australia. CleanUp 2013 (hosted by the CRC for Contamination Assessment and Remediation of the Environment CRC CARE) in Melbourne (presentation by B. Goldsworthy).

Schoof R, Kaden D and Harris A. 2013. Hydraulic fracturing: Human health concerns. Science Advisory Board for Contaminated Sites Third Annual Conference on Contaminated Sites. Vancouver, BC, Canada.

Kaden DA, Harris A, Imse J, Travers M and Schoof R. 2012. Exposure and toxicity of chemicals resulting from natural gas extraction and fracturing. Poster presented at the 51st Meeting of the Society of Toxicology in San Francisco, California.

Johnson D, Schoof R, Moore T, Harris C. 2012. Arsenic and lead biomonitoring studies as tools to assess long-term remedy protectiveness for former mining communities. International Society for Exposure Science in Seattle, WA (presentation by D. Johnson).

Feldpausch A and Schoof R. 2012. Development of a residence-specific, health-based screening criterion for benzo(a)pyrene in settled house dust. International Society for Exposure Science in Seattle, WA (poster).

Spalt E, Johnson D, Schoof R, Baker C and Tobin S. 2012. Tiered approach for chemical prioritization at a former pesticide testing site. International Society for Exposure Science in Seattle, WA (poster).

Schoof R, Moore T, Harris C and Johnson D. 2012. The increasing importance of biomonitoring data to interpret changing risk estimates for legacy mining communities. USEPA Hardrock Mining Conference 2012: Advancing Solutions for a New Legacy. Denver, CO. Presentation.

Schoof RA, Zieber P and Tolbert L. 2009. Probabilistic risk assessment of incremental risk between site and background arsenic in soil. Toxicol Sci 108(S-1) Abstract 844.

Schoof R and Lorenzen E. 2009. Probabilistic lead risk assessment for a community with an operating smelter. Toxicol Sci 108(S-1) Abstract 843.

Cárdenas A, Schoof R, Stupakoff I, Christy A, Cheney D. 2007. Risk Evaluation for Consumption of Cadmium in Pacific Northwest Oysters and International Maximum Limits. Poster presentation at Pacific Northwest Chapter of Society of Environmental Toxicology and Chemistry Conference, April 2007, Port Townsend, WA. DOI: 10.13140/RG.2.2.27784.21762.

Bradley A, Cárdenas A, Curl c, and Schoof R. 2007. Developing and utilizing questionnaire data in building an exposure model for lead. Poster presentation at International Society for Exposure Analysis Annual Conference, Durham, NC.

Zieber P, Cárdenas A, Schoof R and Ostrom T. 2006. Traditional tribal lifeways exposure scenario for an oiled beach in the Pacific Northwest. Poster presented at the Toxics in Puget Sound Forum, April 5, and the Pacific Northwest Society of Environmental Toxicology and Chemistry Conference, April 13–15, Port Townsend, Washington.

Lowney YL, Ruby MV, Wester RC, Schoof RA, Holm SE and Maibach HI. 2005. Percutaneous absorption of arsenic from environmental media. Toxicol Sci 84(S-1) Abstract 2084:427.

Schoof RA, Williams L and Yager JW. 2004. Arsenic speciation and bioaccumulation studies support higher ambient water quality criterion (AWQC). Toxicol Sci 78 (S-1) Abstract 728: 150

Williams L, Schoof RA, Schuler A, Zieber P, Yager JW and Goodrich-Mahoney J. 2004. Arsenic bioaccumulation—implications of using a power function to estimate bioaccumulation factors. Society of Environmental Toxicology and Chemistry, Portland, Oregon.

Kester GB, Brobst RB, Carpenter A, Chaney RL, Rubin AB, Schoof RA and Taylor D. 2004. Risk Characterization, Assessment, and Management of Organic Pollutants in Beneficially Used Residual Products. Sustainable Land Application Conference, Lake Buena Vista, Florida.

Schoof RA. and Yost LJ. 2002. Estimation of inorganic arsenic intake from fish: Market basket vs. recreational catches. Toxicol Sci 66 (1-S) Abstract 1698: 347.

Yost LJ, Schoof RA and Garry M. 2002. Estimation of dietary intake of inorganic arsenic in children. Toxicol Sci 66 (1-S) Abstract 1696: 346.

Schoof RA and Tsuji JS. 2000. The role of outdoor dust in exposures to chemicals in soil: Case studies for arsenic. Toxicol Sci 54 (1-S) Abstract 1168: 249.

Tsuji J, Schoof R and Hook G. 2000. Subchronic health effect levels for childhood exposure to arsenic. Toxicol Sci 54 (1-S) Abstract 346: 73.

Ruby MV, Schoof RA and Carpenter MJ. 1999. Arsenic bioaccessibility and mineralogy from soils at a pesticide manufacturing facility, 15th Annual Conference on Contaminated Soils and Sediments, Amherst, Massachusetts.

Menzel DB, Dillencourt MB, Meacher DM, Lee E, Bic LF, Schoof RA, Yost LJ, Farr CH and Cragin DW. 1999. Monte Carlo analysis of inorganic arsenic exposure in the US Toxicol Sci 48 (1-S) Abstract 1650:350.

Evans CG and Schoof RA. 1998. Soil screening levels for cadmium should account for adult exposures and background exposures. Toxicol Sci 42 (1-S) Abstract 205:41.

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ATTACHMENT 2
IEUBK AND ADULT LEAD MODEL MODELING APPROACH

IEUBK AND ADULT LEAD MODEL MODELING APPROACH

The United States Environmental Protection Agency's (USEPA's) Integrated Exposure Uptake Biokinetic (IEUBK) model and Adult Lead Model (ALM) were used to estimate blood lead levels (BLLs) associated with potential environmental exposures to lead in Lake Tahoe for children and adults, respectively.

In Superfund risk assessments for lead, the IEUBK model is used to predict the risk, as a probability, that a typical child (0 to 6 years old) will have a BLL greater than a specific cut-off value when exposed to a combination of specified media concentrations of lead (USEPA 1994). The model also estimates the distribution of BLLs in the hypothetical population exposed to lead and estimates the geometric mean BLL in the population. The model includes three modules. The exposure module calculates media-specific lead intake rates to estimate how much lead is taken into a child's body from air (indoor and outdoor), soil, dust (indoor), diet, maternal blood lead, and other sources if specified. The uptake model incorporates absorption factors to estimate the fraction of lead intake that crosses into the bloodstream from the lungs or gastrointestinal tract. The transfer of lead between blood and other body tissues and through elimination pathways is addressed by the biokinetic module. The model incorporates numerous default input values and recommends the use of site-specific data where doing so would more accurately predict child blood lead levels. The most recent version of the model, IEUBK v2.0, was validated by Brown et al. (2022). The Brown et al. (2022) validation study found that IEUBK was able to predict BLLs within 0.3 µg/dL of real world geometric mean BLLs. Thus, BLLs less than 0.3 µg/dL are within the range of model error.

Default parameters included in IEUBK v2.0 were used in this assessment, unless otherwise specified (see Table 4A and Table 4B). Because IEUBK was designed to focus on residential exposure to lead in soil, it does not include a default outdoor soil lead concentration. The arithmetic mean lead concentration across all U.S. residential soils as reported in the American Healthy Homes Survey II (106 mg/kg; HUD 2021) was used as the soil lead concentration in this assessment. Using the default IEUBK background exposures and 106 mg/kg as the soil lead concentration, IEUBK predicts a geometric mean BLL of 1.740 µg/dL.

Impacts to BLLs associated with recreational exposure to lead in sediment, surface water and consumption of fish in Lake Tahoe were evaluated using the alternate source menu in IEUBK. Lead intakes from each medium were assessed separately due to differences in absorption/bioavailability. Lead intakes calculated in the exposure assessment for a young child were input in the alternate source menu. IEUBK was then run using the model defaults (plus 106 mg/kg soil concentration) along with the alternate source intake from Lake Tahoe. The geometric mean BLL predicted by IEUBK with the Tahoe-specific source included was compared with the geometric mean BLL predicted by IEUBK using only the model defaults and background soil. If the difference was less than 0.3 µg/dL, the impact to BLL from the Lake Tahoe exposure was considered to have no measurable impact, because it was within the range of model error.

A similar approach was used in the ALM to evaluate impacts to geometric mean BLLs associated with Lake Tahoe lead intakes for adults. The ALM evaluates lead exposure in

pregnant adults and can be used to predict BLLs in both the pregnant adult and the fetus. For consistency with the young child evaluation using IEUBK, the geometric mean BLL predicted by ALM for pregnant adults with and without Tahoe-specific intakes were compared[10]. Unlike IEUBK, ALM does not use media-specific inputs to account for cumulative lead exposure. Instead, it incorporates a baseline BLL of 0.6 µg/dL (the geometric mean BLL for women ages 17 – 45 from the 2009 - 2014 National Health and Nutrition Examination Survey) which includes background exposure to lead in food, drinking water, and residential soil and dust. Environmental lead exposures in the ALM are focused on additional soil and dust exposures occurring outside the home, typically assumed to occur at a workplace. A soil lead concentration of zero should be entered if there is no additional soil source of lead exposure. Thus, a background or 'default' run of the ALM (assuming a soil lead concentration of zero) yields the baseline BLL (0.6 µg/dL) as the geometric mean BLL for adults.

The ALM is an Excel spreadsheet and the formulas can be edited to incorporate additional lead exposures. The ALM formula estimating adult geometric mean BLL was edited to account for Tahoe-specific intakes from surface water (recreational exposure), sediment (recreational exposure), and fish in accordance with USEPA's recommendation for evaluating dietary exposures in the ALM Frequently Asked Questions webpage (USEPA 2024). The BLLs predicted by ALM using the Tahoe-specific exposures were compared with the baseline BLL (0.6 µg/dL). There are no readily available validation studies for the ALM. So, it is unclear what predicted values are within the range of model error. The geometric mean BLLs predicted in all the Tahoe model runs were virtually indistinguishable from the baseline BLL (i.e., < 0.001 µg/dL). Changes in BLL < 0.001 µg/dL are not scientifically meaningful, so the Tahoe-specific intakes were considered to have no measurable increase to average BLLs.

<u>Drinking water exposure</u>

Because drinking water exposure is already included in the IEUBK model, the approach for evaluating lead exposure from Lake Tahoe water when used as drinking water differs slightly from the other exposure scenarios. The lead concentration selected to represent Tahoe surface water used as drinking water was simply used as the drinking water concentration in the IEUBK water menu, and the predicted geometric mean BLL was compared to the IEUBK default. Predicted geometric mean BLLs within 0.3 µg/dL were considered within the range of model error.

The baseline BLL incorporated in the ALM accounts for background drinking water exposure. Thus, incorporating drinking water exposure from another source would cause this pathway to be double-counted. There is no USEPA guidance related to the evaluation of drinking water exposure using ALM. Because of this issue, drinking water exposures were not evaluated for adults. Lead concentrations in Tahoe surface water near the cables (0.012 µg/L) are lower than average lead concentrations (0.9 – 1 µg/L) in the United States drinking water supply. The IEUBK evaluation for children found no measurable difference in Tahoe vs IEUBK default assumptions for BLLs when background lead in drinking water is assessed. Adults were also assumed to have no impact to BLLs if Tahoe water was consumed as drinking water.

[10] The ALM does not predict geometric mean BLLs for fetuses. It predicts the 95th percentile BLL among fetuses of pregnant adults and the probability that fetal BLL exceeds a target BLL (USEPA 2017).

Table A2-1. IEUBK Model Input Parameters

Parameter	Value	Basis
Soil concentration (mg/kg)	106 mg/kg	Arithmetic mean soil lead concentration, all residential housing units from American Healthy Homes Survey II (HUD 2021).
Dust concentration (mg/kg)	C_{dust} = 0.7 • $C_{soil(weighted)}$ + (air concentration • 100)	Derived from residential soil and air concentrations
Outdoor air concentration (µg per cubic meter [m^3])	0.1	IEUBK default
Indoor air concentration (µg/m^3)	30% of outdoor air concentration	IEUBK default
Drinking water concentration (µg per liter [L])	0.9	IEUBK default
Maternal blood lead at birth (µg/dL)*	0.6	IEUBK default
Absorption fractions at low intakes: Air Diet Water Soil/dust	 32% 50% 50% 30%	 IEUBK default IEUBK default IEUBK default IEUBK default
Relative Bioavailability (RBA; soil/dust)	60%	IEUBK default
Fraction soil	45%	IEUBK default
Geometric Standard Deviation (GSD)	1.6	IEUBK default

Table A2-2. Age-Dependent Input Parameters to the IEUBK Model[1]

Age (months)[2]	Air		Diet	Water	Soil-Dust
	Time Outdoors (hours)	Ventilation Rate (m^3/day)[3]	Dietary Intake (µg/day)	Intake (L/day)	Incidental Ingestion Rate (mg/day)[4]
0 to <12	1.0	3.22	2.66	0.4	86
12 to <24	2.0	4.97	5.03	0.43	94
24 to <36	3.0	6.09	5.21	0.51	67
36 to <48	4.0	6.95	5.38	0.54	63
48 to <60	4.0	7.68	5.64	0.57	67
60 to <72	4.0	8.32	6.04	0.6	52

Notes

[1] Values shown are IEUBK defaults.

[2] The recommended age range for Superfund sites using the IEUBK model is 12–72 months.

[3] The values shown are the midpoint of the age range.

[4] Soil-dust ingestion rate in the IEUBK model is apportioned as follows: 45% intake is soil and 55% intake is dust.

Table A2-3. Adult Lead Model Input Parameters[1]

Variable	Description of Variable	Units	Value
PbS	Soil lead concentration	µg/g or ppm	0
$R_{fetal/maternal}$	Fetal/maternal PbB ratio	--	0.9
BKSF	Biokinetic Slope Factor	µg/dL per µg/day	0.4
GSD_i	Geometric standard deviation PbB	--	1.8
PbB_0	Baseline PbB	µg/dL	0.6
IR_S	Soil ingestion rate (including soil-derived indoor dust)	g/day	0.050
$AF_{S,D}$	Absorption fraction (same for soil and dust)	--	0.12
$EF_{S,D}$	Exposure frequency (same for soil and dust)	days/yr	219
$AT_{S,D}$	Averaging time (same for soil and dust)	days/yr	365

Notes

[1] Values shown are Adult Lead Model defaults.

REFERENCES – ATTACHMENT 2

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